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Report Number III

The Effects of Blast Trauma (Impulse Noise)
on Hearing: A Parametric Study
Source II

Report No. ARL 90-2

Annual Report

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There are three broad goals to this contract. The first and primary goal is to study the effects of high level blast wave exposure on the conductive and sensory structures of the mammalian ear. This includes the use of the auditory evoked potential to measure hearing thresholds and tuning curves prior to and after exposure to various blast wave exposures. Parameters of the blast waves studied include intensity, spectral composition, number of impulses and repetition rate. Correlations among hearing measures, exposure variables and histology have been developed. To achieve the above, the following two objectives must be completed: (1) develop a series of blast wave generation devices which are suitable for the laboratory simulation of a wide spectrum of blast waves; and develop a suitable set of software and a PC-based computer system which will interact with crystal and capacitive microphones to capture and analyze blast waves. Four sources have been developed along with the specified analysis system. The results from the Source I (a conventional shock tube) were summaried in reports ADA 206-180 and ADA 203-854. This report summaries the results from Source II, a 5-inch Lamont valve-driven shock tube. (KR)

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SUMMARY

There are three broad goals to this project. The primary goal is to begin the systematic development of a data base from which one could estimate the hazards to hearing resulting from exposure to blast waves or other high level impulse noise transients. To achieve this objective the following two additional objectives must first be achieved: (1) develop a methodology to efficiently acquire data on a large number of experimental animals that have been exposed to a variety of blast wave configurations. This includes audiometric, histological and acoustic variables; (2) develop a set of blast wave simulation devices which can reliably generate blast waves with a variable distribution of spectral energy in a laboratory environment.

Two previous progress reports (ADA 206-180 and ADA 203-854) from this contract have documented the results and methodology associated with items (1) and (2) above. In addition, the audiometric and histological results from 109 chinchillas exposed to very low frequency non-reverberant blast waves produced by Source I were described. This present report is written in two sections. The first section documents the results of parametric experiments performed on 105 chinchillas exposed to the mid-frequency blast waves produced by the 5-inch Lamont source (Source II). The second section presents a comparison of the spectral effects between Source I and II. The objective of these experiments was to correlate the exposure variables with functional and morphological indices of trauma. The following blast wave parameters were studied: 1. Intensity of the blast wave. The intensity was characterized by the peak sound pressure level, and by the total energy (J/m^2) of the exposure. Three intensities were used: 150, 155, and 160 dB peak SPL. 2. Inter-stimulus interval (ISI) i.e., the effects of repetition rate. Rates of 1/m, 10/m, and 1/10m were used. 3. Total number of impulses (N), where N = 1, 10, and 100.

The conclusions that can be drawn from these data are identical to those that were made for the impulses produced by Source I (report ADA 206-180). However, the two sources do differ in the magnitude of the absolute energy levels of the exposure at which trauma begins to develop. In summary, (1) There was no statistical difference in the amount of hearing loss or the amount of sensory cell loss for exposure to a single impulse at 150, 155, or 160 dB peak SPL. Individual animals showed no permanent hearing loss and no significant sensory cell loss. (2) The variability in hearing and cell losses across animals increases as the severity of the exposure increases. This increase in variability seems to be tied more to peak levels of the stimulus than it is to the total energy. The variability in the results makes it difficult to describe the data with conventional statistics. (3) A general, though not surprising, trend in the data is that as the peak levels and the N increase permanent effects increase; these permanent effects seem to be dependent upon peak levels more than upon the total energy in the exposure stimulus. Also, for a constant peak and energy level, the more rapid presentation rate (10/min) generally seemed to produce the greater effect although the effect is not consistent.

The second section of this report focuses on a comparative analysis of Source I and II and the effect that the impulse spectrum has on the extent of hearing trauma. Although this analysis is considered preliminary, there do appear to be consistent trends evolving. Specifically, for all frequencies even those for which there is relatively little stimulus (impulse) energy, exposures to Source I are less traumatic than are exposures to Source II. This difference between sources is frequency dependent and varies from approximately 0 to 5 dB.

FOREWORD

<u>Disclaimer</u>:

Citations of commercial organizations and trade names in this report do not constitute an official Department of the Army endorsement or approval of the products or services of these organizations.

<u>Animal Use</u>:

In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals," prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council (DHHS Publication No. (NIH) 86-23, revised 1985).

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I. INTRODUCTION

There are a number of different suggested standards for exposure to impulse/impact noise [e.g. Coles, et al. (1968), Smoorenburg (1982), and Pfander (1980)]. Although each of these criteria has its proponents, there is a consensus that there is, in fact, an extremely limited empirical data base upon which a standard can be built. The difficulties associated with generating a data base are compounded by the extremely broad range of high intensity noise transients that exist in various industrial and military environments. For example, in industry, impacts often occur as a pseudorandom sequence, having variable peak intensities, and are superimposed on a continuous noise background. This combination produces a highly non-Gaussian noise of variable character often with a very high kurtosis. While rms SPLs' might be within the limits of hearing conservation standards, peaks in excess of 130 dB or more can be very common but irregular in their temporal characteristics. At the other extreme, the diversity of military weapon systems produce impulses which originate as the result of a process of shock wave formation and propagation following high energy discharges. These waves, which can have peak levels in excess of 180 dB, can be either reverberant or non-reverberant in nature depending upon the environment in which they are encountered and they also may be superimposed on a background noise. Trying to develop a single standard to cover this broad range of "acoustic" signals is a formidable task.

The primary goal of this research project is to produce a data base from which one could estimate the hazards to hearing associated with a wide variety of non-reverberant blast wave exposures. To achieve this objective four different blast wave generation devices were designed. Three of these sources are based upon shock tube methods and one uses a high energy electrical discharge to produce a shock wave. The four sources produce pressure-time waveforms whose A-weighted amplitude spectra peak at four different regions of the audible spectrum. The conventional shock tube (Source I) has maximum A-weighted energy in the .250 kHz octave band; the 5-inch "Lamont" rapid acting valve driven shock tube (Source II) has its energy maxima in the 1 kHz octave band; the 2-inch "Lamont" tube (Source III) has its energy maxima in the 2 kHz octave band; while the spark discharge energy (Source IV) is concentrated at the 4 kHz octave band. These sources in anechoic surroundings produce non-reverberant waves that approximate the ideal Friedlander wave. By varying the exposure variables such as peak sound pressure level (SPL), number of impulses and the presentation rate, the relation between these variables and auditory system trauma can be established. A brief background and literature review which summarizes the current state of knowledge on the contribution of these parameters to hearing loss is presented in the first progress report ADA 206-180 which documents the results of exposure to the very low frequency blast waves that are produced by Source I. This present report, which documents the audiometric and histological results of exposure to the impulses produced by Source II, follows a pattern of data presentation that parallels the presentation format of report ADA 206-180. In addition to the importance of the parameters mentioned above, when the data collection from all four sources is complete the relation between the spectral characteristics of the impulse and the subsequent hearing loss can begin to be explored. This spectral question is an important one for which very little experimental data are available. The second part of this report focuses upon the question of spectral sensitivity by performing a preliminary analysis of the data base formed by Sources 1 and II.

II. BACKGROUND

One of the surprising features of the existing or proposed exposure criteria is the general lack of specific consideration that is given to the frequency domain representation of the impulse, a point frequently raised by Price (1983) and others. Some deference is, however, given to the spectrum in these criteria, but in a rather covert or indirect manner; e.g., through the use of A-weighting of the stimulus or through the handling of the A and B duration variables. The authors of the Coles, et al. (1968) proposal, for example, acknowledge the importance of the spectrum by recognizing that long A-duration Friedlander type waves, which transport relatively large amounts of low frequency energy, are less effective in producing acoustic trauma than are short duration A-waves. Thus, their criterion line for the "A-waves" is drawn horizontal for A-durations greater than approximately 1 ms. A more direct spectral approach to the evaluation of impulses and impacts was proposed by Kryter (1970). His suggestions, while based upon sound reasoning, never really caught on. The Kryter approach appeared attractive in its ability to predict the amount of temporary threshold shift measured two minutes after exposure (TTS_2) to a noise transient provided that the TTS_2 was not very large or alternatively that the levels of the transient in any given frequency band were not excessive. Price (1983, 1986) to some extent has tried to build upon and extend the Kryter approach by considering the spectral transmission characteristics of the peripheral auditory system. Price's reasoning led to the following conclusions. (1) There is a species specific frequency, f_o, at which the cochlea is most vulnerable and that impulses whose spectrum peaks at f_o will be most damaging. This would appear to be true, according to Price, regardless of the distribution of energy above and below f_o. For man the suggested frequency is 3.0 kHz. (2) Relative to the threshold for damage at f_o, the threshold for damage should rise at 6 dB/octave for $f_p < f_o$ and at 18 dB/octave for $f_p > f_o$ where f_p = spectral peak of the impulse. Thus, a model for permanent damage was developed which is amenable to experimental testing. In subsequent studies Price (1983) has tried to relate, with varying degrees of success, experimental data obtained from the cat to the predictions of this model.

A review of the literature indicates that, except for the Price studies, there are few published results obtained from experiments specifically designed to study the effects of the spectrum of an impulse on hearing trauma. When the data collection from all four blast wave sources is completed we will have a data base from approximately 450 animals from which the relation between spectrum and auditory trauma can be formulated.

III: PARAMETRIC EXPERIMENTS

A. EXPERIMENTAL METHODS

The methodology used to acquire the data presented in this report has been reported in detail in our earlier report ADA 206-180. Briefly, the basic experimental protocol that is common to all of the experiments consists of the following steps: (1) Preexposure audiograms and tuning curves (TC's) are measured on each animal. (2) The animals are exposed to noise under well controlled conditions. The temporal and spectral characteristics of the noise are recorded. (3) The animal's evoked response thresholds are again measured immediately after exposure and at regular intervals after exposure. At 30 days postexposure, the audiogram is again measured to establish the animal's permanent threshold shift, (PTS), and postexposure TC's are once again collected at all audiometric test frequencies. (4) The animals are

euthanatized and their cochleas are then prepared for microscopic analysis. Cochleograms, which provide a quantitative description of the extent and location of the hair cell lesions, are prepared for each cochlea.

Subjects: The chinchilla was used as the experimental animal. Over the years, the chinchilla has been used in a wide variety of auditory experiments and consequently, much is known about its threshold (Miller, 1970; Salvi et al., 1978), psychophysical tuning curves (McGee et al., 1976; Salvi et al., 1982a), threshold for gap detection (Giraudi et al., 1980) and amplitude modulated noise (Salvi et al., 1982b). These psychophysical results indicate that the chinchilla's hearing capabilities are quite similar to those of man. The chinchilla is perhaps the most common animal used in noise trauma research even though there is a general consensus that the species is more susceptible to noise trauma than is man. However, phenomenologically the chinchilla is considered to be a suitable model for man. Thus, the chinchilla was chosen as a reasonable animal model for the blast wave studies described in this report.

One hundred and five (105) chinchillas were used in this study. Each animal was anesthetized [IM injection of Ketamine (12.86 mg/kg), Acepromazine (0.43 mg/kg) and Xylazine (2.57 mg/kg)] and made monaural by the surgical destruction of the left cochlea. A chronic electrode was implanted near the inferior colliculus for single-ended near-field recording of the evoked potential (Henderson et al., 1973; Salvi et al., 1982a). The animals were allowed to recover for at least a week before evoked potential testing began.

Preexposure testing: Hearing thresholds were estimated on each animal using the auditory evoked potential (AEP). The AEP has been shown to be a valid index of hearing threshold in the chinchilla. The correlation between the behavioral and evoked response measures has been strengthened by directly comparing, in the same animal, estimates of noise-induced behavioral and evoked potential threshold shifts (Henderson et al., 1983; Davis and Ferraro, 1984). There is a close correlation between the behavioral and evoked response thresholds before, during, and after acoustic overstimulation. In other words, the evoked potential threshold estimation procedure provides a good estimate of the magnitude of noise-induced hearing loss. The animals were awake during testing and restrained in a yoke-like apparatus to maintain the animal's head in a constant position within the calibrated sound field. AEP's were collected to 20 msec tone bursts (5 msec rise/fall time) presented at a rate of 10 per second. A general-purpose computer (Digital Equipment Corporation MicroPDP-11/73) with 12-bit A/D converter(Data Translation 3362), timer (ADAC 1601) and digital interface (ADAC 1632) was used to acquire the evoked potential data and control the frequency, intensity and timing of the stimulus via a programmable oscillator (Wavetek 5100), programmable attenuator (Spectrum Scientific MAT) and electronic switch (Coulbourn Instruments S84-04). The electrical signal from the implanted electrode was amplified (50,000x) and filtered (30 Hz to 3000 Hz) by a Grass P511J biological amplifier and led to the input of the A/D converter where it was sampled at 20 kHz (50 msec period) over 500 points to obtain a 25 msec sampling window. Each sampled waveform was analyzed for large amplitude artifacts; and if present, the sample was rejected from the average and another sample taken. Averaged AEP's were obtained from 250 presentations of the 20 msec signal. Each waveform was stored on disk for later analysis.

Thresholds were measured using an intensity series with 5 dB steps at octave intervals from 0.5 to 16.0 kHz and at the half-octave frequency of 11.2 kHz. Threshold was determined to be one half step size (2.5 dB) below the lowest intensity that showed a "response" consistent with the responses seen at higher intensities. The intensity resolution of our method is 5 dB. The

average of at least three separate threshold determinations at each frequency obtained on different days was used to obtain the preexposure audiogram.

Tone-on-tone masking functions (i.e., AEP tuning curves, see e.g., Salvi et al., 1982a) were measured on each animal at six probe frequencies between 0.5 and 11.2 kHz presented at 15 dB above the preexposure threshold. A simultaneous masking paradigm was used (McGee et al., 1976). The probe tone had a duration of 20 ms and the intensity was set at 15 dB sensation level at the given test frequency. A simultaneous pure tone masker was presented at increasing levels until the masker just abolished the evoked potential elicited by the probe tone. The procedure was repeated over a range of masker frequencies around the probe tone to yield a "V" shaped masking function. The AEP has been shown to provide as good an estimate of the frequency selectivity as that obtained by behavioral techniques (Salvi et al., 1982a). It also shows that a small population of neurons within a restricted frequency band are contributing to the AEP at near threshold intensities. The advantage of the AEP tuning curves is that they provide an independent method of assessing frequency selectivity and a method that is much easier to apply than behavioral techniques. Ten masker frequencies (from a Wavetek Model 23 programmable frequency synthesizer) distributed in frequency above and below the probe tone frequency were presented in an intensity series with 5 dB steps. The masked threshold was taken as one half a step size (2.5 dB) above the last masker intensity that resulted in a "response". TC's were run on 63 chinchillas (i.e., 3 from each group) from which 378 preexposure TC's and 378 postexposure TC's were obtained. All the individual animal data is tabulated in a three volume data appendix which will be available upon request at the termination of this contract. The results of the analysis of TC data is published in Davis et al. (1989) and will not be repeated in this document.

Blast Wave Generation, Measurement and Analysis: A principal requirement for this study was the precise measurement and recording of the blast wave. The computer system used for this purpose was a Compaq 286 Deskpro personal computer using the ASYST™ application package (ASYST™ Software Technologies, Inc., Rochester, NY). The blast wave was first digitized and then recorded in storage devices (e.g., hard disk or magnetic tape). By using the customized software developed in our laboratory, each digitized blast wave was analyzed to extract characteristics such as the total acoustic energy, energy spectrum, peak and root-mean-square (RMS) sound pressure level (SPL) etc.

A schematic representation of the blast wave exposure test facility using the 5-inch "Lamont" source is illustrated in Figure 1. A cross-sectional view of the "Lamont" driver is shown in Figure 2. The Lamont source uses a relatively simple rapid acting valve to quickly establish a high pressure discontinuity in the expansion section in order to "drive" the shock front. A force differential generated over the area of the low pressure chamber relative to the high pressure chamber, on the rear plate, maintains the seal of the high pressure chamber. As the low pressure is gradually reduced a point is reached where the net force acting on the value reverses direction and the valve rapidly thrusts forward releasing the "slug" of high pressure gas into the expansion section. N_2 is used as the operating gas and the pressure in the high pressure chamber varies from approximately 100 psig to 1000 psig to achieve peak sound pressure levels of the blast wave of from 150 dB to 160 dB at the exposure location. The SPL of the blast wave can be controlled by systematically adjusting the pressure in the compression section. The pressure-time history of the blast wave was recorded using a transducer located on the center line at a variable distance from the outlet
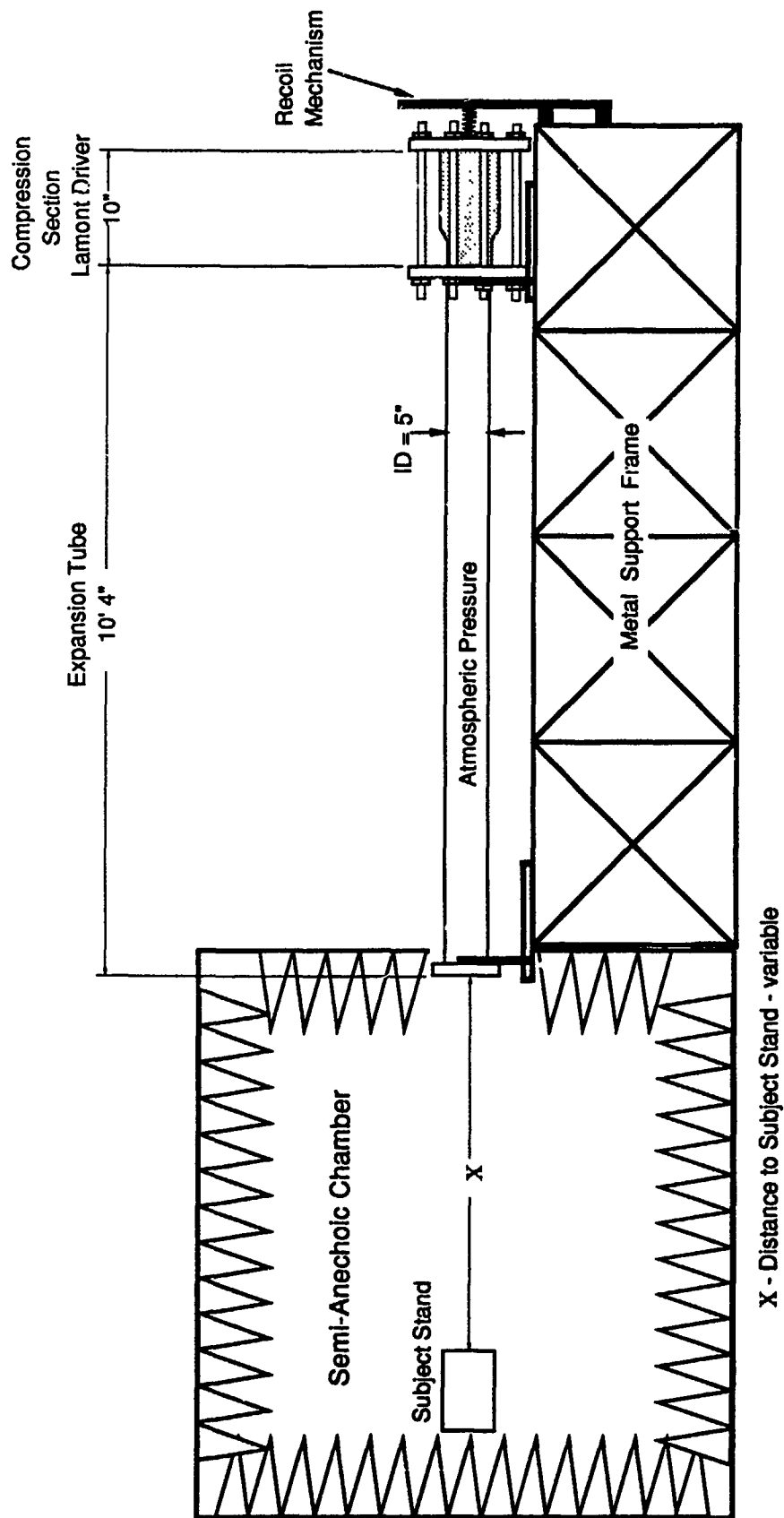
Figure 1. Schematic Side View of the Lamont Shock Tube

X - Distance to Subject Stand - variable
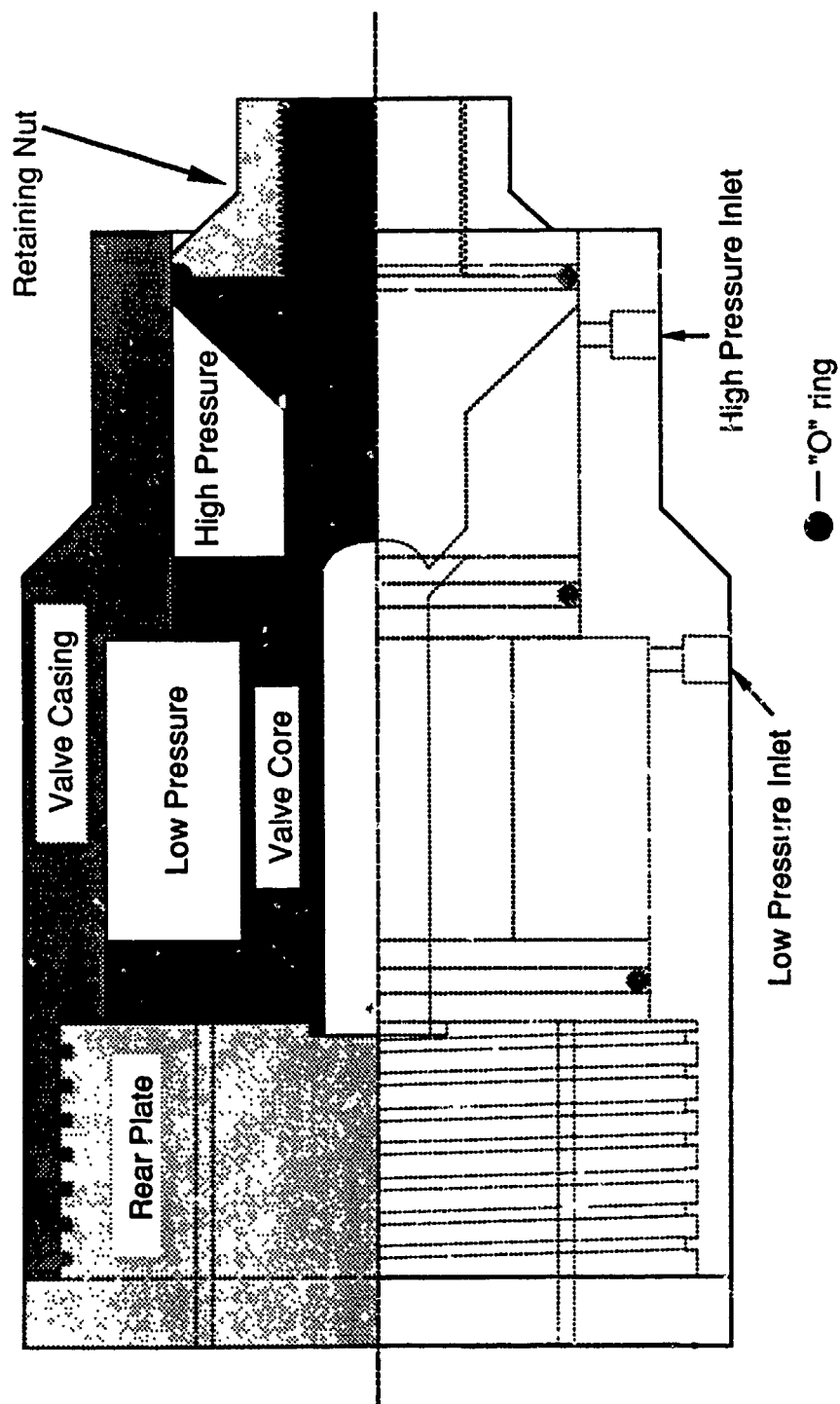
Figure 2. Schematic of the Lamont Quick-acting Valve

Retaining Nut

High Pressure

Valve Casing

Low Pressure

Valve Core

Rear Plate

High Pressure Inlet

Low Pressure Inlet

●—"O" ring

of the shock tube. The experimental animal was mounted next to the transducer.

Two different types of transducers were used to convert the dynamic acoustic pressure into an analog signal. The B&K 1/8 inch microphone (Type 4138) and the PCB crystal microphone (Model 112A22) were selected because of their ability to record high peak levels and their relatively fast rise times. A B&K microphone preamplifier (Type 2639), a B&K measuring amplifier (Type 2606), and a PCB six-channel amplifying power unit (Model 483A08) were used to amplify the analog signals from the B&K and PCB microphones respectively. Both transducers yielded identical results. The amplified analog signals were monitored on an oscilloscope. The output signal from the transducers was amplified and, in order to avoid aliasing problems that can occur in analog-to-digital (A/D) conversion, the amplified signals were filtered using an anti-aliasing filter prior to digitizing. The sampling rate of the A/D convertor (12-bit) was set at 500 kHz and the cut off frequency of the anti-aliasing filter was set at 150 kHz (approximately 1/3 of the sampling rate). For each blast wave, 16,384 samples were recorded for later analysis. Software was written using this PC-based system to perform the following tasks. total sound exposure and exposure level calculations (Young, 1970); energy flux calculations; and spectral analysis using a 4096-point FFT; A-weighted analysis, etc.

Thus, for each impact the total sound exposure or exposure level could be calculated (i.e., the time integrated, squared sound pressure). For the impulse data presented here, the total sound exposure was divided by the standard characteristic impedance of air, $\rho c = 406$ mks rayls, to produce a quantity with units of energy flux (i.e., J/m^2). Similarly, all spectral quantities $|P(\omega)|^2$ were converted to units of energy flux spectral density, and for each impulse exposure, the total "energy flux" in the octave bands having center frequencies at the audiometric test frequencies was calculated. [Since only p(t) was measured, the true energy flux cannot be obtained except in the special case of a plane wave.]

Exposure of Animals: For a given exposure condition, each chinchilla was exposed at the same fixed location relative to the horn exit. During exposure the animal was unanesthetized but immobilized in a leather harness (Patterson et al., 1986). The right pinna was folded back and fixed in place to insure that the entrance of the external meatus was not obstructed and the position of the entire animal was adjusted so that the cross sectional plane of the meatus was oriented parallel to the advancing shock front (i.e., a normal incidence).

Each experimental group of animals generally consisted of five animals. Each animal was individually exposed to one of the exposure conditions shown in Table I. A total of 105 animals were used to complete this experimental paradigm.

Postexposure Testing: After the exposure was complete, threshold recovery functions were measured at 0.5, 2.0 and 8.0 kHz at 0, 2, 8, 24 and 240 hours after removal from the noise (using the same method as described for preexposure testing). After at least 30 days, final audiograms were constructed using the average of three separate threshold determinations at each of the seven preexposure frequencies. Permanent threshold shift (PTS) was defined as the difference between the postexposure and preexposure thresholds at each individual test frequency. Postexposure AEP tuning curves were collected at the six preexposure probe tones presented at 15 dB above the postexposure threshold.

TABLE I

A Definition of the Experimental Groups

Group	N	Intensity	Number	Rate
1	5	150 dB Peak SPL	1	
2	5	150 dB Peak SPL	10	10 per minute
3	5	150 dB Peak SPL	10	1 per minute
4	5	150 dB Peak SPL	10	1 per 10 minutes
5	5	150 dB Peak SPL	100	10 per minute
6	5	150 dB Peak SPL	100	1 per minute
7	5	150 dB Peak SPL	100	1 per 10 minutes
8	5	155 dB Peak SPL	1	
9	5	155 dB Peak SPL	10	10 per minute
10	5	155 dB Peak SPL	10	1 per minute
11	5	155 dB Peak SPL	10	1 per 10 minutes
12	5	155 dB Peak SPL	100	10 per minute
13	5	155 dB Peak SPL	100	1 per minute
14	5	155 dB Peak SPL	100	1 per 10 minutes
15	5	160 dB Peak SPL	1	
16	5	160 dB Peak SPL	10	10 per minute
17	5	160 dB Peak SPL	10	1 per minute
18	5	160 dB Peak SPL	10	1 per 10 minutes
19	5	160 dB Peak SPL	100	10 per minute
20	5	160 dB Peak SPL	100	1 per minute
21	5	160 dB Peak SPL	100	1 per 10 minutes

Total 105

Cochlear Histology: Following postexposure audiometric testing, animals were euthanatized by decapitation and the cochleas were immediately removed and fixed. The cochleas were dissected and the status of the sensory cell population was evaluated using conventional surface preparation histology (Engstrom et al., 1966). Briefly, the stapes was removed and the round window membrane opened to allow transcochlear perfusion, via the scala tympani/scala vestibuli with cold 2.5% glutaraldehyde in veronal acetate buffer at 7.3 pH (605 mOsm). Postfixation was performed on the following day with one percent osmium tetroxide in veronal acetate buffer (pH 7.3) for 30 minutes. The cochleas were dissected and the entire sensory epithelium along with the lateral wall structures was mounted in glycerin on glass slides. [See Hamernik et al., (1987) for a more complete description]. The status of sensory and supporting cells were evaluated with Nomarski Differential Interference Contrast microscopy and entered into a data-base on a laboratory computer (Digital Equipment Corporation MicroPDP-11/73 and Macintosh II). Standard cochleograms were then constructed by computing the percent sensory cell loss across the length of the cochlea in 0.24 mm steps. These cell loss figures were then converted into percent loss over octave bands centered at the audiometric test frequencies along the length of the cochlea and correlated with the frequency-place map constructed by Eldredge et al. (1981).

B. RESULTS

The results of the present experiments are grouped into sections devoted to preexposure threshold data, analysis of the exposure stimuli and postexposure threshold and histological data. The audiometric and histological dependent variables were analyzed using mixed design analyses of variance with repeated measures on one factor (frequency). The SPSS^X statistical package was used and the probability of a type I error was set at 0.05.

Preexposure Thresholds: The mean preexposure thresholds for all 105 animals are reported in Table II and plotted in Figure 3 along with the behavioral audibility curve published by Miller (1970). The Miller curve was corrected for the effects of temporal integration using the data of Henderson, (1969). The error bars in this figure represent one standard deviation above and below the mean.

Table II

Summary of Mean Preexposure Thresholds (dB) for
All Animals (N = 105) Compared to Published Norms

	Test Frequency (kHz)							
Present study	0.5	1.0	2.0	4.0	8.0	11.2	16.0	
	21.4	4.2	7.7	-0.3	14.2	14.6	22.0	$\bar{X}$
	6.0	5.8	6.1	7.6	7.1	8.3	9.6	s
Miller (1970)	5.1	3.0	2.7	1.9	5.8	9.9	12.1	$\bar{X}$
(750 ms signals)	6.1	4.1	4.7	7.1	5.4	6.7	6.9	s
	36	36	36	36	36	34	36	N
Miller (1970) corrected for temporal integration (Henderson, 1969)	16.2	14.1	13.8	13.0	16.9	21.0	23.2	$\bar{X}$
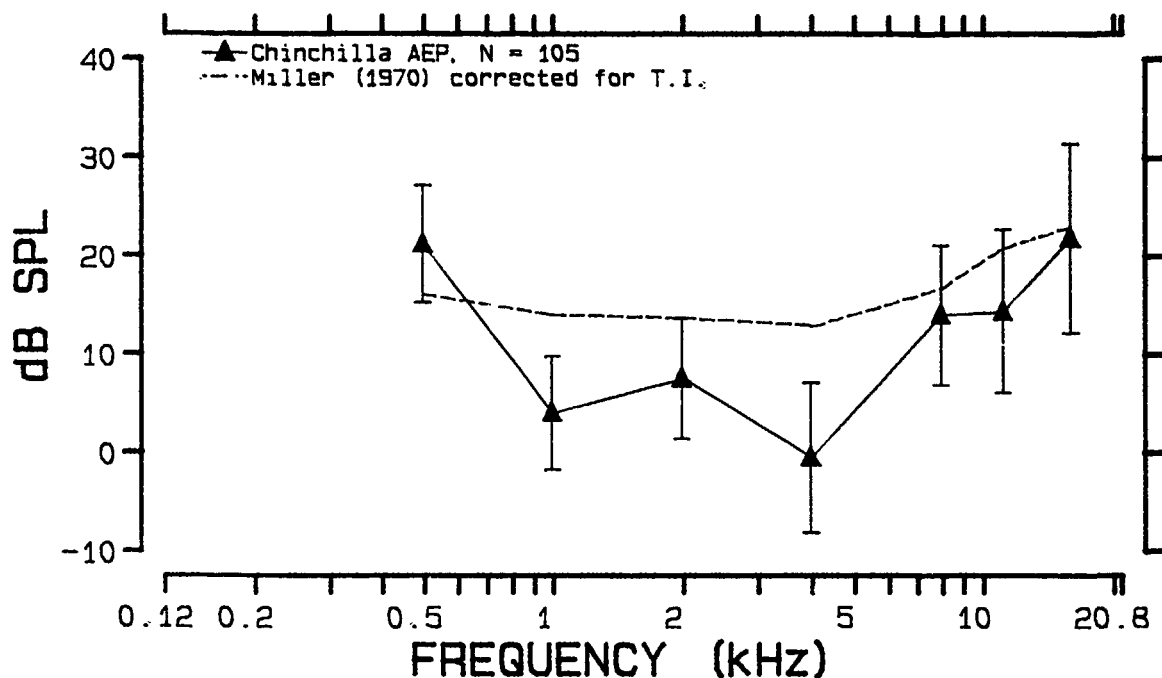
Figure 3. Mean Preexposure Thresholds for 105 chinchillas.

The mean preexposure thresholds are generally better than Miller's (1970) behavioral thresholds at the mid-frequencies when the (approximate 11.1 dB) effects of temporal integration are taken into consideration. Lower thresholds, which are also found in other published data, probably reflect improvements in the techniques of AEP recording. The mean preexposure thresholds for the 21 individual groups of animals and the mean preexposure thresholds for all 105 subjects are summarized in Table III.

The audiological dependent variables in this report are maximum threshold shift (TS_{max}) and permanent threshold shift (PTS). Each of these variables is computed by subtracting the preexposure from the postexposure thresholds. Thus, each animal serves as its own control subject. There were no statistically significant differences in mean preexposure thresholds among groups (F = 1.43, df = 20/84). There was a statistically significant frequency main effect (F = 205.26, df = 6/504) that was anticipated on the basis of our previous knowledge of the chinchilla audiogram (Fay, 1988). The interaction between group and frequency was not statistically significant (F = 1.10, df = 120/504). The analysis of variance for the preexposure thresholds is summarized in Table IV.

Noise Exposures: Pressure-time histories for each of the three intensity waves produced by Source II are shown in Figures 4 (a-c). A time record over a period of 10 ms is shown. The Fourier energy spectrum for these same three waveforms over the entire 33 ms window is also shown in Figure 4 (a-c). The total energy flux for each exposure condition is presented in Table V in relative dB levels as well as in Joules/meter^2. A tabulation of the octave band A-weighted and unweighted energy flux values for a single impulse at 150, 155 and 160 dB peak SPL is presented in Table VI. The data presented in Table V is also shown plotted as a bar graph in Figure 5 to facilitate the comparison of the three waves used for the exposures. For all three peak intensities, each wave had a similar p-t profile and similar A-weighted and unweighted spectral distribution of energy. The A-weighted

TABLE III

Preexposure Threshold Means (dB) and Standard Deviations for all Groups

dB Peak	#	Rate	N	0.5	1.0	2.0	4.0	8.0	11.2	16.0	
150 dB	1		5	21.5	4.2	6.8	1.8	16.2	14.8	19.5	$\bar{X}$
				3.3	5.3	3.8	7.7	4.2	11.8	8.1	s
150 dB	10	10/M	5	24.5	8.8	11.2	2.8	21.5	18.5	23.2	$\bar{X}$
				3.6	10.0	11.3	3.8	4.8	7.6	6.7	s
150 dB	10	1/M	5	21.5	2.8	7.5	-1.8	11.2	15.8	17.2	$\bar{X}$
				6.0	9.1	3.9	6.9	4.5	4.1	6.9	s
150 dB	10	1/10M	5	19.8	3.8	6.2	2.8	16.5	13.8	22.5	$\bar{X}$
				3.7	2.7	5.6	6.5	5.8	6.3	8.7	s
150 dB	100	10/M	5	24.8	4.5	7.8	-3.2	10.5	13.2	15.8	$\bar{X}$
				3.8	3.0	5.1	2.2	7.0	5.7	9.1	s
150 dB	100	1/M	5	22.2	3.2	4.2	1.2	10.5	6.8	15.2	$\bar{X}$
				3.0	2.8	4.3	9.2	10.4	8.8	9.6	s
150 dB	100	1/10M	5	20.5	3.2	5.8	-3.2	12.5	14.2	18.5	$\bar{X}$
				6.2	4.3	10.0	5.3	8.8	10.1	8.5	s
155 dB	1		5	20.5	5.2	9.8	-3.2	12.2	15.5	21.8	$\bar{X}$
				6.3	3.0	4.9	8.1	6.8	3.6	12.1	s
155 dB	10	10/M	5	16.8	1.5	5.8	-2.5	13.5	10.2	21.2	$\bar{X}$
				3.3	1.9	4.6	4.6	5.8	3.3	9.2	s
155 dB	10	1/M	5	23.5	3.2	8.2	-1.8	12.2	12.2	17.5	$\bar{X}$
				3.7	1.9	6.9	4.5	8.9	4.0	8.3	s
155 dB	10	1/10M	5	23.8	6.2	7.8	1.5	18.8	11.8	21.5	$\bar{X}$
				10.6	6.3	3.2	7.0	8.5	6.4	10.7	s
155 dB	100	10/M	5	25.2	5.8	7.5	-3.5	15.8	8.5	20.5	$\bar{X}$
				8.2	3.7	5.7	6.5	4.9	11.9	9.1	s
155 dB	100	1/M	5	21.8	3.8	9.5	2.5	12.2	15.8	18.5	$\bar{X}$
				3.8	2.2	8.2	5.4	12.1	13.8	12.2	s
155 dB	100	1/10M	5	22.2	6.8	9.5	2.2	15.5	9.8	24.5	$\bar{X}$
				9.1	5.8	4.3	8.4	11.6	11.9	13.0	s
160 dB	1		5	19.2	0.8	1.5	-3.2	11.5	14.8	25.2	$\bar{X}$
				4.1	7.6	8.5	7.4	4.0	4.7	12.1	s
160 dB	10	10/M	5	24.3	0.6	7.0	-0.4	16.7	14.5	26.4	$\bar{X}$
				9.2	8.0	3.4	4.6	3.4	6.2	8.9	s
160 dB	10	1/M	5	17.5	3.5	7.2	-2.5	12.2	14.8	22.2	$\bar{X}$
				3.1	2.5	4.6	3.1	6.4	5.3	6.1	s
160 dB	10	1/10M	5	16.5	0.5	10.8	3.8	14.8	18.5	30.2	$\bar{X}$
				2.2	5.3	4.9	9.1	1.9	7.2	12.2	s
160 dB	100	10/M	5	16.5	-0.5	6.5	-10.5	9.5	12.5	25.8	$\bar{X}$
				3.3	4.2	3.3	5.9	9.1	6.6	9.6	s
160 dB	100	1/M	5	21.5	8.5	12.8	4.8	19.2	24.2	22.5	$\bar{X}$
				10.2	8.1	3.2	11.3	5.3	5.1	7.0	s
160 dB	100	1/10M	5	25.2	12.5	8.8	5.5	15.5	25.8	32.5	$\bar{X}$
				8.3	5.7	10.4	15.3	4.3	6.8	8.6	s

Table IV

Analysis of Variance Summary Table of Preexposure Thresholds

Source of Variation	SS	df	MS	F	p
Group	4036.69	20	201.83	1.43	.131
Between Subjects	11839.05	84	140.94		
Frequency	45168.88	6	7528.15	205.27	.000
Group x Frequency	4848.42	120	40.40	1.10	.239
Within Subjects	18484.28	504	36.68		

Table V

Total Weighted and Unweighted Energy Flux (J/m^2) Values for Each Exposure Condition

Peak SPL (dB)	Weight	Absolute Energy (J/m^2)			Relative Energy (dB) re: $1J/m^2$		
		1X	10X	100X	1X	10X	100X
150	None	0.14	2.54	13.90	-8.54	4.05	11.43
	A	0.08	1.49	7.79	-10.97	1.73	8.92
155	None	0.45	4.51	45.09	-3.47	6.54	16.54
	A	0.30	2.98	29.77	-5.23	4.74	14.74
160	None	1.14	11.42	114.15	0.57	10.58	20.57
	A	0.72	7.17	71.69	-1.43	8.56	18.55

Table VI

Octave Band Unweighted and A-Weighted Energy Flux (J/m^2) for a Single Impulse Generated by the 5" Lamont Shock Tube.

| Octave Band CF (kHz)	150 dB Peak SPL		155 dB Peak SPL		160 dB Peak SPL	
	Unwtg. Energy	A-Wtg. Energy	Unwtg. Energy	A-Wtg. Energy	Unwtg. Energy	A-Wtg. Energy
< 0.125	0.0197	0.0000	0.0474	0.0159	0.1553	0.0549
0.125	0.0036	0.0001	0.0177	0.0007	0.0588	0.0024
0.25	0.0369	0.0039	0.0890	0.0105	0.2193	0.0309
0.5	0.0168	0.0075	0.0773	0.0382	0.1876	0.0954
1.0	0.0219	0.0213	0.1073	0.1034	0.2603	0.2530
2.0	0.0136	0.0179	0.0495	0.0650	0.0845	0.1115
4.0	0.0142	0.0180	0.0339	0.0427	0.0751	0.0944
8.0	0.0040	0.0030	0.0096	0.0078	0.0474	0.0370
16.0	0.0035	0.0012	0.0099	0.0042	0.0274	0.0114
> 16.0	0.0050	0.0050	0.0092	0.0092	0.0260	0.0260
Total	0.1390	0.0779	0.4509	0.2977	1.1415	0.7169

Source II
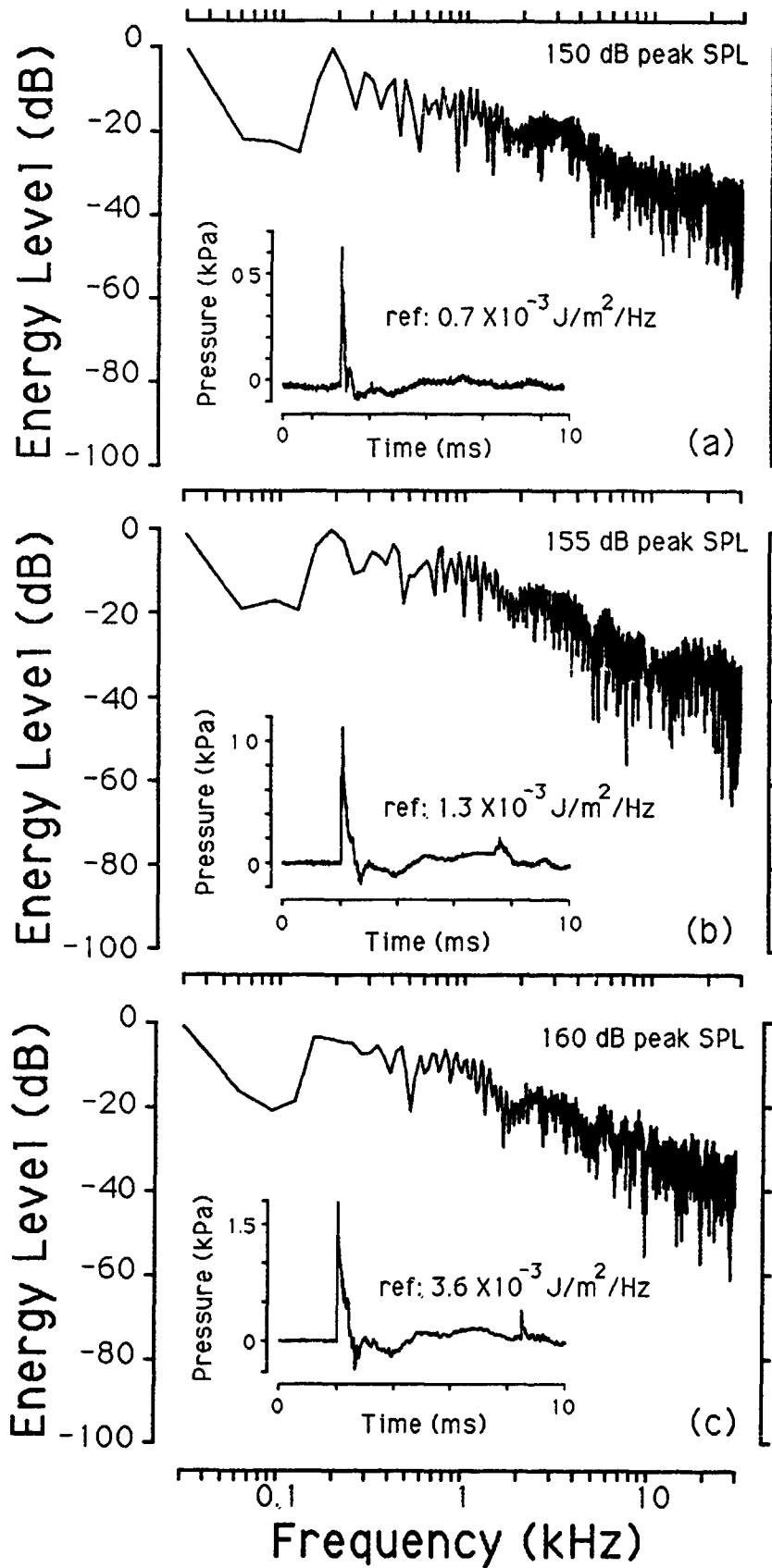
Figure 4.   Amplitude spectra and pressure-time waveforms for the blast waves
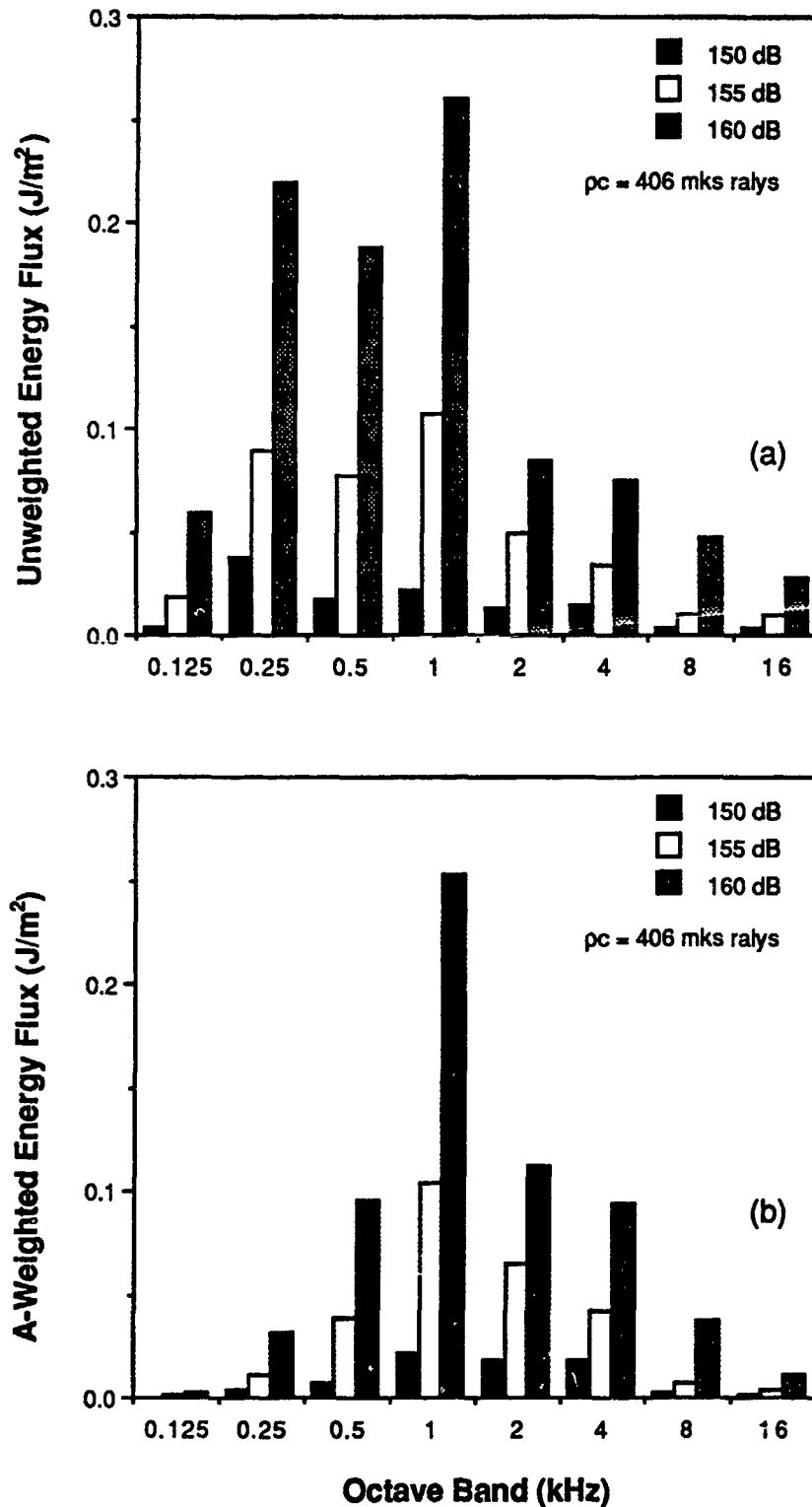from the 5" Lamont shock tube.

5" Lamont Shock Tube

Figure 5. The unweighted (upper) and A-weighted (lower) octave band analysis of the three blast waves.

analysis shows that the peak of the energy spectrum lies in the 1 kHz octave band for each of the three intensity waves.

Postexposure Audiometric Results: There are four independent variables in the present experiments: number of impulses (1X, 10X or 100X), impulse peak level (150, 155 or 160 dB SPL), impulse presentation rate (10/min, 1/min or 1/10m), and frequency (i.e., audiometric test frequency or basilar membrane location). The dependent variables are maximum threshold shift (TS_{max}), permanent threshold shift (PTS), percent outer hair cell loss and percent inner hair cell loss. The independent variable of frequency is the only within-subjects variable. The experimental design did not lend itself to a typical four factor mixed design analysis of variance since there was no rate variable for the three groups exposed to a single impulse. Therefore, several different analyses were performed on each of the four dependent variables. Since the rate variable could not be applied to an analysis which included all groups, the rate variable was analyzed as one variable in a three-factor mixed-design analysis of variance with impulse peak and frequency as the other two factors. In other words, two separate three-factor analyses were performed, the first on the groups exposed to 10 impulses, and the second on the groups exposed to 100 impulses (see Tables VIII and IX). The remaining three groups (i.e., the 1x groups) were analyzed using a two-factor mixed-design analysis of variance with only impulse peak and frequency as independent variables (see Table VII). Thus, each of the above analysis were performed only upon those groups that were exposed to an equal number of impulses. The main effect of the number of impulses was determined using a separate three-factor mixed design analysis of variance with peak, number and frequency as the independent variables (i.e., the data were collapsed across rate, see Table X).

Figures 6 through 12 present the audiometric and histological results for 1, 10 and 100 impulses. The error bars represent one standard error of the mean above and below the mean. If error bars are not present, the standard error was less than the size of the symbol representing the mean. The following is an interpretation of the results presented in these figures and tables.

1. Frequency: The main effect of frequency represents the only within-subjects independent variable. In general, the main effect of frequency was statistically significant for most of the analyses that are reported in this manuscript. A significant frequency main effect suggests that the audiometric or histological losses are different at the various audiometric test frequencies or locations along the basilar membrane. The statistically significant interactions of between-subjects independent variables and frequency indicate that the effect of the between-subjects variable depends on the frequency at which the dependent variable is measured. Thus, a statistically significant interaction between peak and frequency for percent outer hair cell loss tells us that the effect of impulse peak on the outer hair cell losses depended upon the frequency (i.e., place on the basilar membrane) that the losses were measured. From examining the figures, it is apparent that little losses were sustained at the locations on the basilar membrane associated with the very low and very high frequencies, while the most severe effects of the impulse were in the mid-frequency region of the cochlea. Since a statistically significant main effect of frequency and interactions between frequency and other main effects are expected in this type of study, we will not discuss frequency effects or interactions at length throughout the remainder of this report. It is important to remember, however, that many of the statistically significant effects interact with the within-subjects frequency variable and thus the significance of the between-

Table VII

Analysis of Variance Summary Table for Groups Exposed to 1 Impulse

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	69.01	2	34.51	.63	.547
Between Subjects	652.22	12	54.35		
Frequency	348.27	2	174.14	4.49	.022
Peak x Frequency	547.65	4	136.91	3.53	.021
Within Subjects	930.00	24	38.75		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	229.58	2	114.79	2.47	.127
Between Subjects	558.10	12	46.51		
Frequency	65.82	6	10.97	.45	.841
Peak x Frequency	205.61	12	17.13	.71	.740
Within Subjects	1745.24	72	24.24		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	2.90	2	1.45	.60	.567
Between Subjects	29.23	12	2.44		
Frequency	6.68	7	.95	.61	.747
Peak x Frequency	36.33	14	2.59	1.65	.081
Within Subjects	131.75	84	1.57		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	18.92	2	9.46	.38	.691
Between Subjects	298.27	12	24.86		
Frequency	935.57	7	133.65	16.07	.000
Peak x Frequency	78.63	14	5.62	.68	.792
Within Subjects	698.60	84	8.32		

Table VIII

Analysis of Variance Summary Table for Groups Exposed to 10 Impulses

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	61776.13	2	30888.06	40.10	.000
Rate	4428.20	2	2214.10	2.87	.069
Peak x Rate	10338.23	4	2584.56	3.36	.020
Between Cells	27731.91	36	770.33		
Frequency	4739.82	2	2369.91	28.98	.000
Peak x Frequency	338.63	4	84.66	1.04	.395
Rate x Frequency	335.02	4	83.76	1.02	.401
Peak x Rate x Frequency	522.51	8	65.31	.80	.606
Within Cells	5888.26	72	81.78		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	8159.81	2	4079.90	11.33	.000
Rate	1317.99	2	659.00	1.83	.175
Peak x Rate	6373.92	4	1593.48	4.43	.005
Between Subjects	12962.94	36	360.08		
Frequency	5600.78	6	933.46	15.61	.000
Peak x Frequency	2646.46	12	220.54	3.69	.000
Rate x Frequency	785.91	12	65.49	1.10	.365
Peak x Rate x Frequency	2123.51	24	88.48	1.48	.076
Within Subjects	12917.34	216	59.80		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	696.60	2	348.30	1.51	.235
Rate	467.63	2	233.81	1.01	.374
Peak x Rate	451.82	4	112.96	.49	.744
Between Subjects	8317.33	36	231.04		
Frequency	1294.24	7	184.89	2.87	.007
Peak x Frequency	905.45	14	64.67	1.01	.449
Rate x Frequency	710.15	14	50.73	.79	.682
Peak x Rate x Frequency	1560.46	28	55.73	.87	.664
Within Subjects	16215.96	252	64.35		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	26572.33	2	13286.16	11.91	.000
Rate	5968.34	2	2984.17	2.68	.083
Peak x Rate	15206.24	4	3801.56	3.41	.018
Between Subjects	40151.90	36	1115.33		
Frequency	49886.87	7	7126.70	25.14	.000
Peak x Frequency	23494.28	14	1678.16	5.92	.000
Rate x Frequency	2630.10	14	187.86	.66	.810
Peak x Rate x Frequency	20790.75	28	742.53	2.62	.000
Within Subjects	71450.24	252	283.53		

Table IX

Analysis of Variance Summary Table for Groups Exposed to 100 Impulses

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	22812.63	2	11406.31	13.18	.000
Rate	3354.43	2	1677.22	1.94	.159
Peak x Rate	2599.33	4	649.83	.75	.564
Between Subjects	31150.20	36	865.28		
Frequency	8612.78	2	4306.39	24.47	.000
Peak x Frequency	371.63	4	92.91	.53	.716
Rate x Frequency	417.53	4	104.38	.59	.669
Peak x Rate x Frequency	1138.12	8	142.27	.81	.598
Within Subjects	12673.55	72	176.02		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	23711.13	2	11855.56	5.58	.008
Rate	10028.69	2	5014.35	2.36	.109
Peak x Rate	2013.74	4	503.43	.24	.916
Between Subjects	76483.33	36	2124.54		
Frequency	1946.84	6	324.47	2.65	.017
Peak x Frequency	2074.06	12	172.84	1.41	.162
Rate x Frequency	1114.64	12	92.89	.76	.693
Peak x Rate x Frequency	1679.22	24	69.97	.57	.948
Within Subjects	26462.22	216	122.51		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	4570.94	2	2285.47	4.85	.014
Rate	1376.70	2	688.35	1.46	.246
Peak x Rate	870.90	4	217.73	.46	.763
Between Subjects	16489.55	35	471.13		
Frequency	3169.76	7	452.82	2.72	.010
Peak x Frequency	3131.18	14	223.66	1.34	.183
Rate x Frequency	1921.33	14	137.24	.82	.643
Peak x Rate x Frequency	1304.48	28	46.59	.28	1.000
Within Subjects	40826.17	245	166.64		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	69487.40	2	34743.70	5.47	.009
Rate	23822.16	2	11911.08	1.88	.168
Peak x Rate	9391.34	4	2347.83	.37	.829
Between Subjects	222330.50	35	6352.30		
Frequency	49563.41	7	7080.49	16.58	.000
Peak x Frequency	19587.37	14	1399.10	3.28	.000
Rate x Frequency	11774.63	14	84.04	1.97	.021
Peak x Rate x Frequency	5827.41	28	20.12	.49	.987
Within Subjects	104614.00	245	427.00		

Table X

Analysis of Variance Summary Table for all Groups Collapsed Across the Rate Variable

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	33556.38	2	16778.19	20.07	.000
Number	75279.61	2	37639.80	45.02	.000
Peak x Number	15196.39	4	3799.10	4.54	.002
Between Subjects	80254.52	96	835.98		
Frequency	6950.13	2	3475.06	30.46	.000
Peak x Frequency	789.08	4	197.27	1.73	.145
Number x Frequency	1391.76	4	347.94	3.05	.018
Peak x Number x Frequency	482.28	8	60.29	.53	.834
Within Subjects	21905.00	192	114.09		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	10549.85	2	5274.93	4.61	.012
Number	22161.52	2	11080.76	9.69	.000
Peak x Number	7586.08	4	1896.52	1.66	.166
Between Subjects	109738.71	96	1143.11		
Frequency	2166.83	6	361.14	4.44	.000
Peak x Frequency	694.14	12	57.85	.71	.741
Number x Frequency	3297.24	12	274.77	3.38	.000
Peak x Number x Frequency	3675.59	24	153.15	1.88	.007
Within Subjects	46828.09	576	81.30		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	1769.45	2	884.73	3.00	.055
Number	3897.10	2	1948.55	6.61	.002
Peak x Number	1459.76	4	364.94	1.24	.300
Between Subjects	28025.20	95	295.00		
Frequency	1231.57	7	175.94	1.87	.072
Peak x Frequency	858.57	14	61.33	.65	.823
Number x Frequency	1800.42	14	128.60	1.36	.165
Peak x Number x Frequency	2181.83	28	77.92	.83	.723
Within Subjects	62664.41	665	94.23		

Percent Outer Hair Cell Loss

| Source of Variation	SS	df	MS	F	p
Peak	35943.84	2	17971.92	5.41	.006
Number	69222.02	2	34611.01	10.41	.000
Peak x Number	17344.92	4	4336.23	1.30	.274
Between Subjects	315806.01	95	3324.27		
Frequency	32520.60	7	4645.80	14.20	.000
Peak x Frequency	11316.39	14	808.31	2.47	.002
Number x Frequency	26319.38	14	1879.96	5.75	.000
Peak x Number x Frequency	17214.85	28	614.82	1.88	.004
Within Subjects	217569.00	665	327.17		
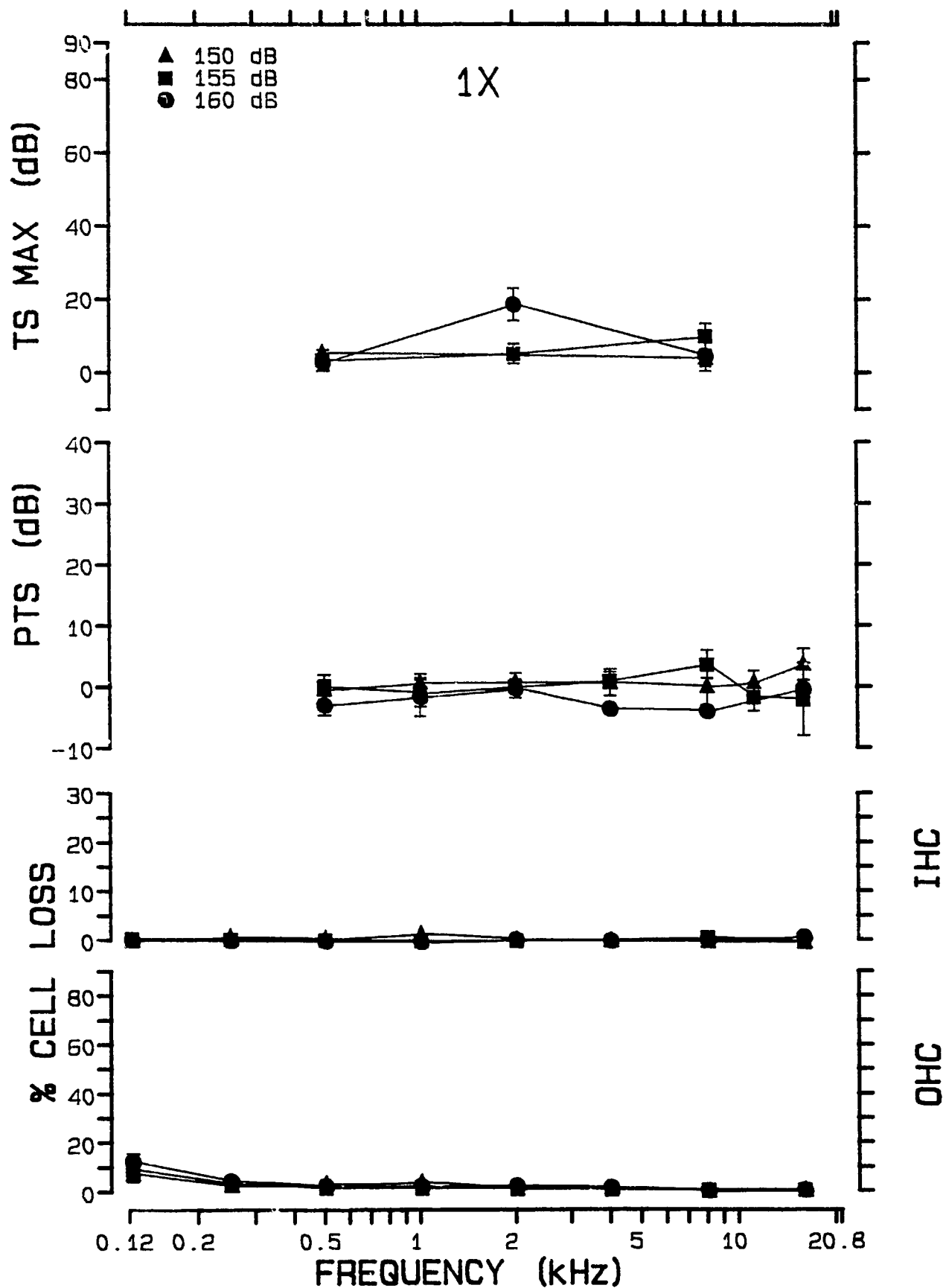
Figure 6. Mean TS$_{max}$ (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 1 blast wave.
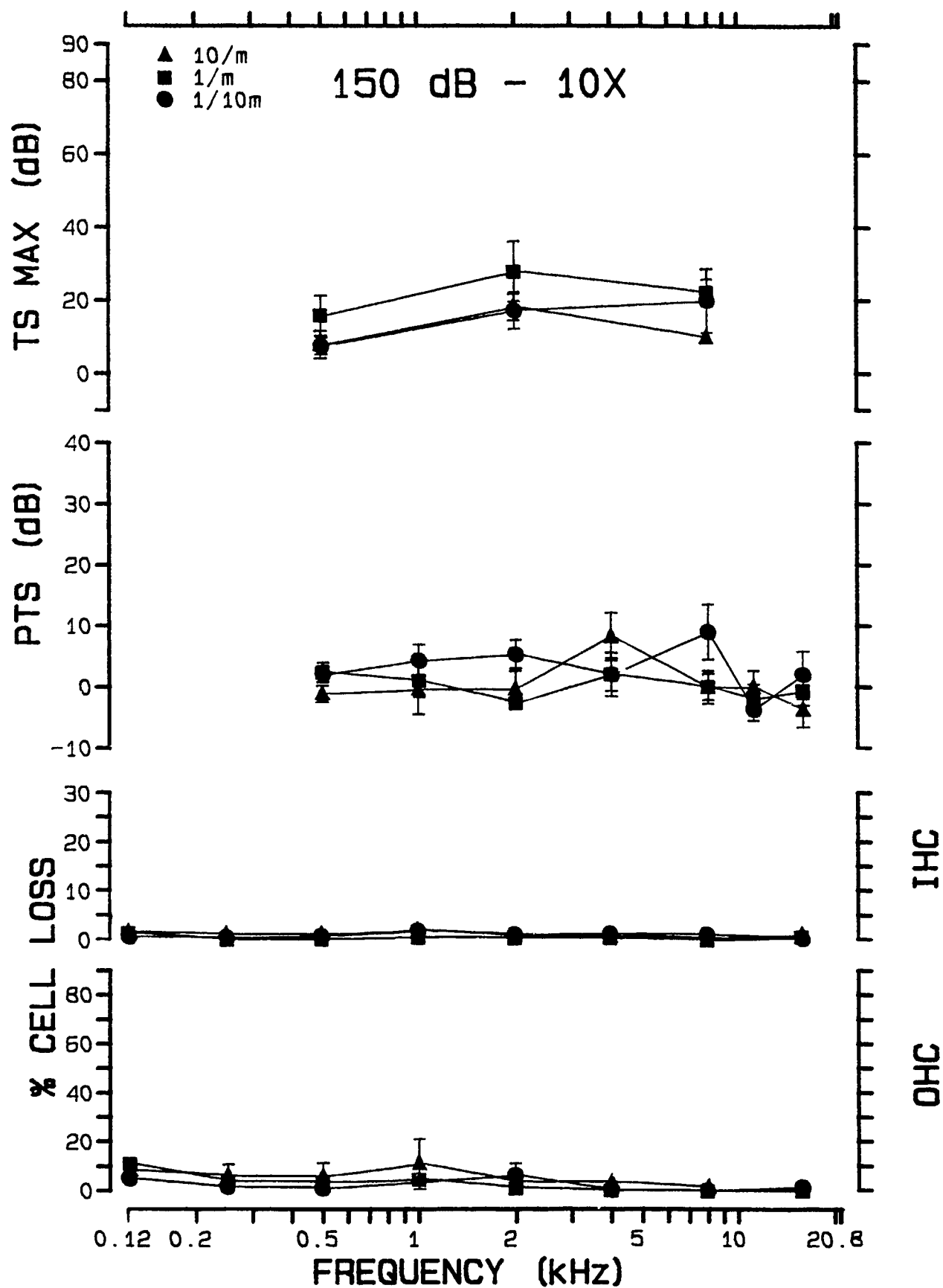
Figure 7. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 150 dB peak SPL.
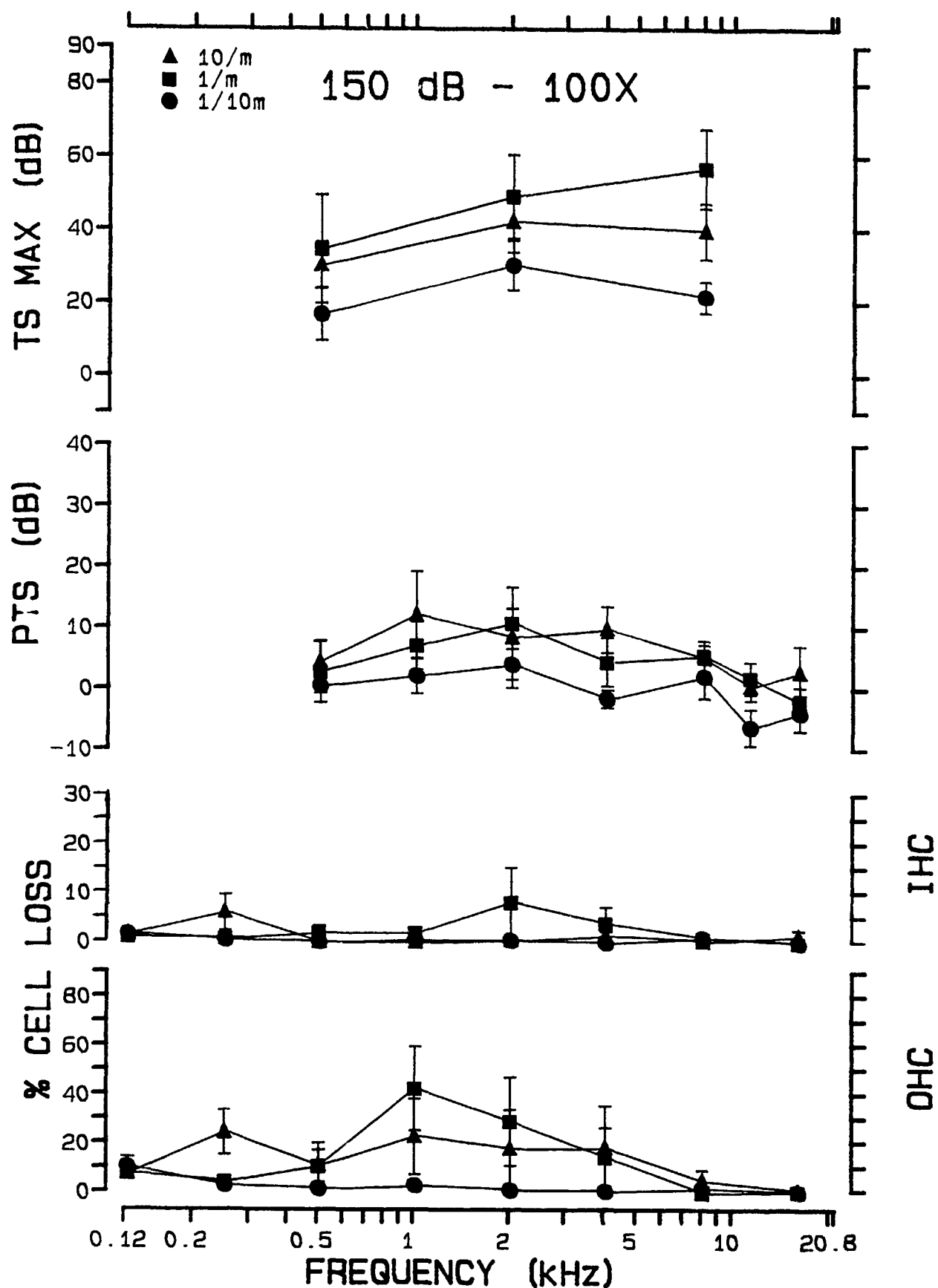
Figure 8. Mean TS$_{max}$ (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 150 dB peak SPL.
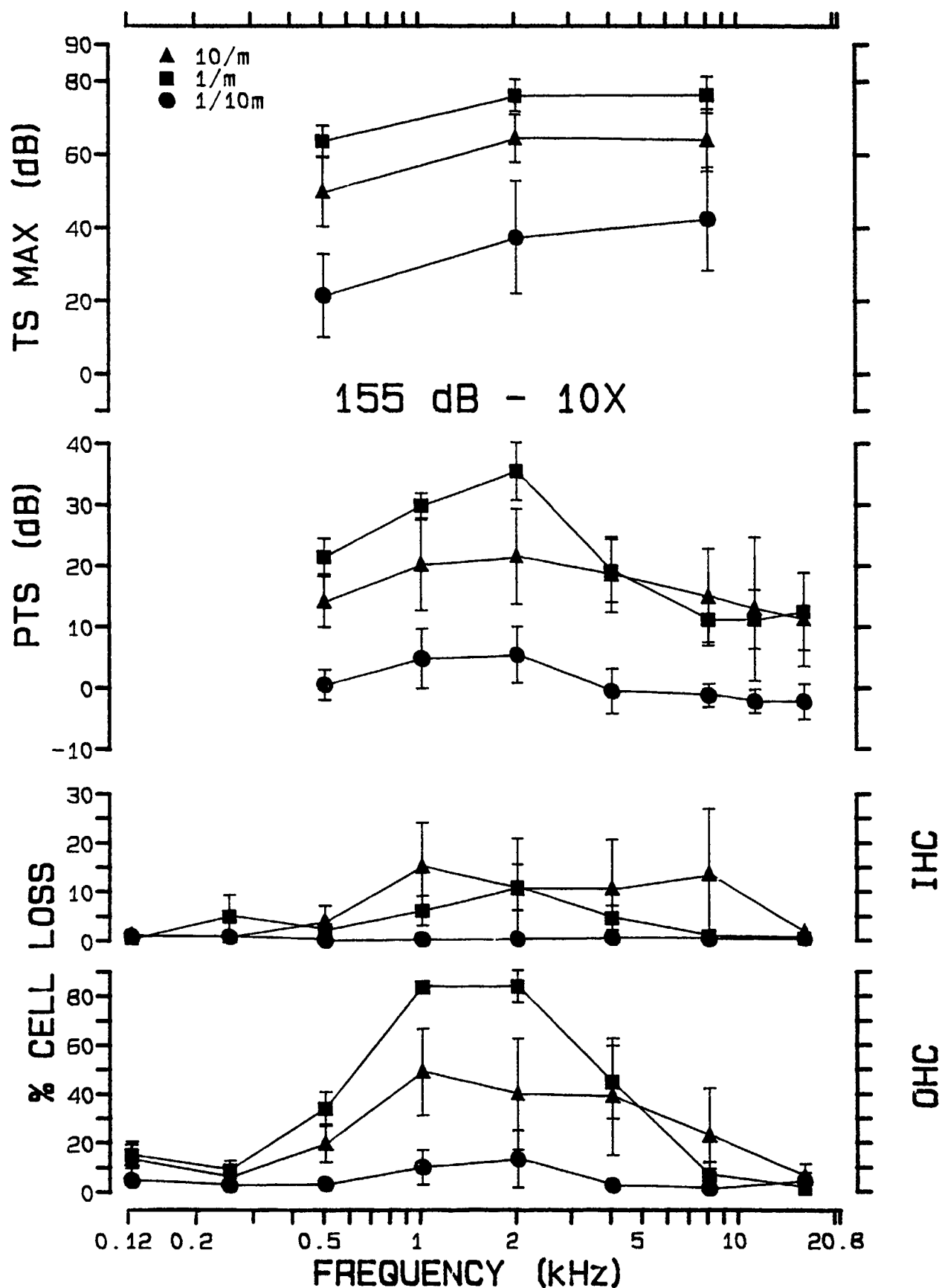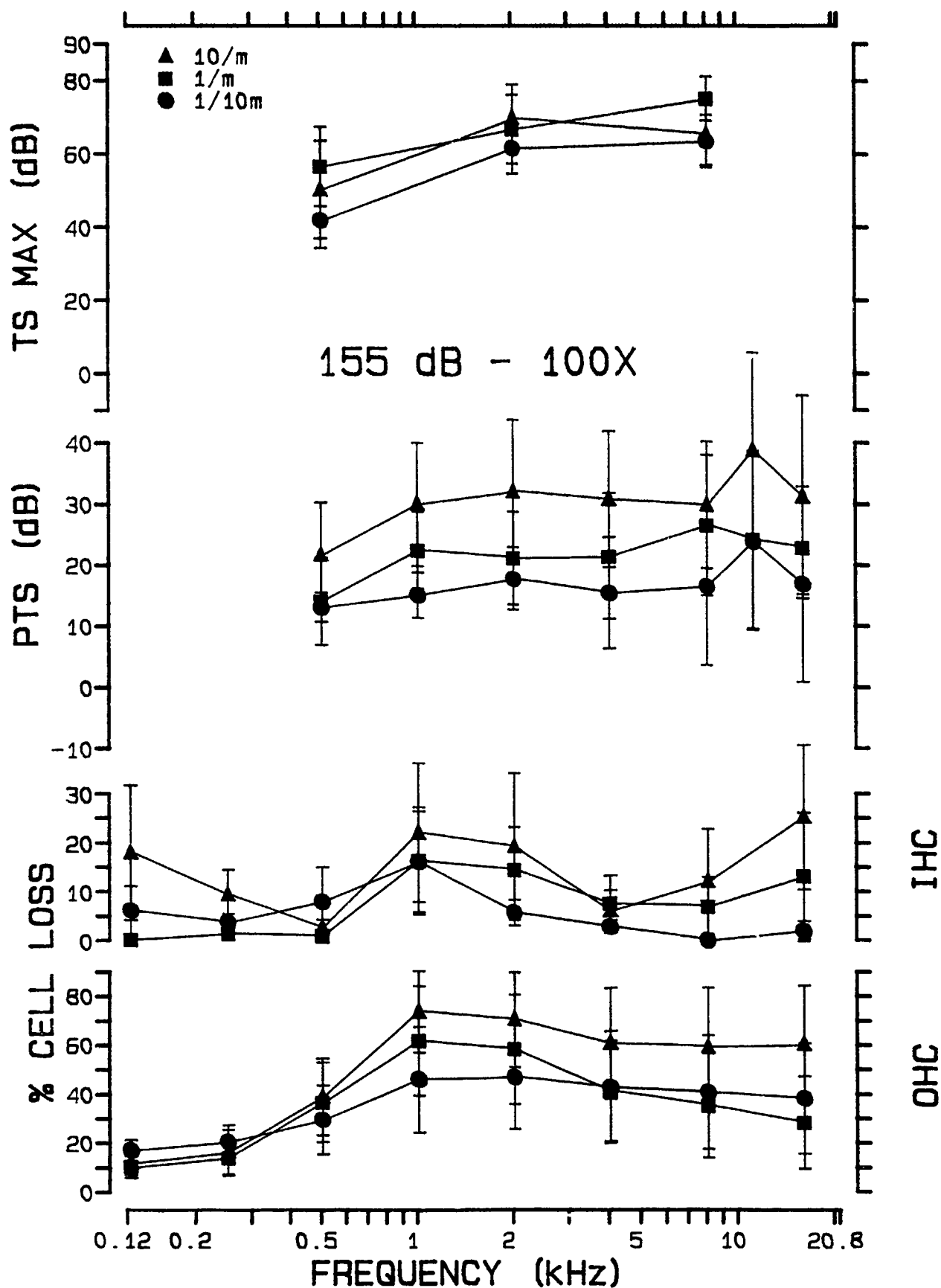
Figure 9. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 155 dB peak SPL.

Figure 10. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 155 dB peak SPL.
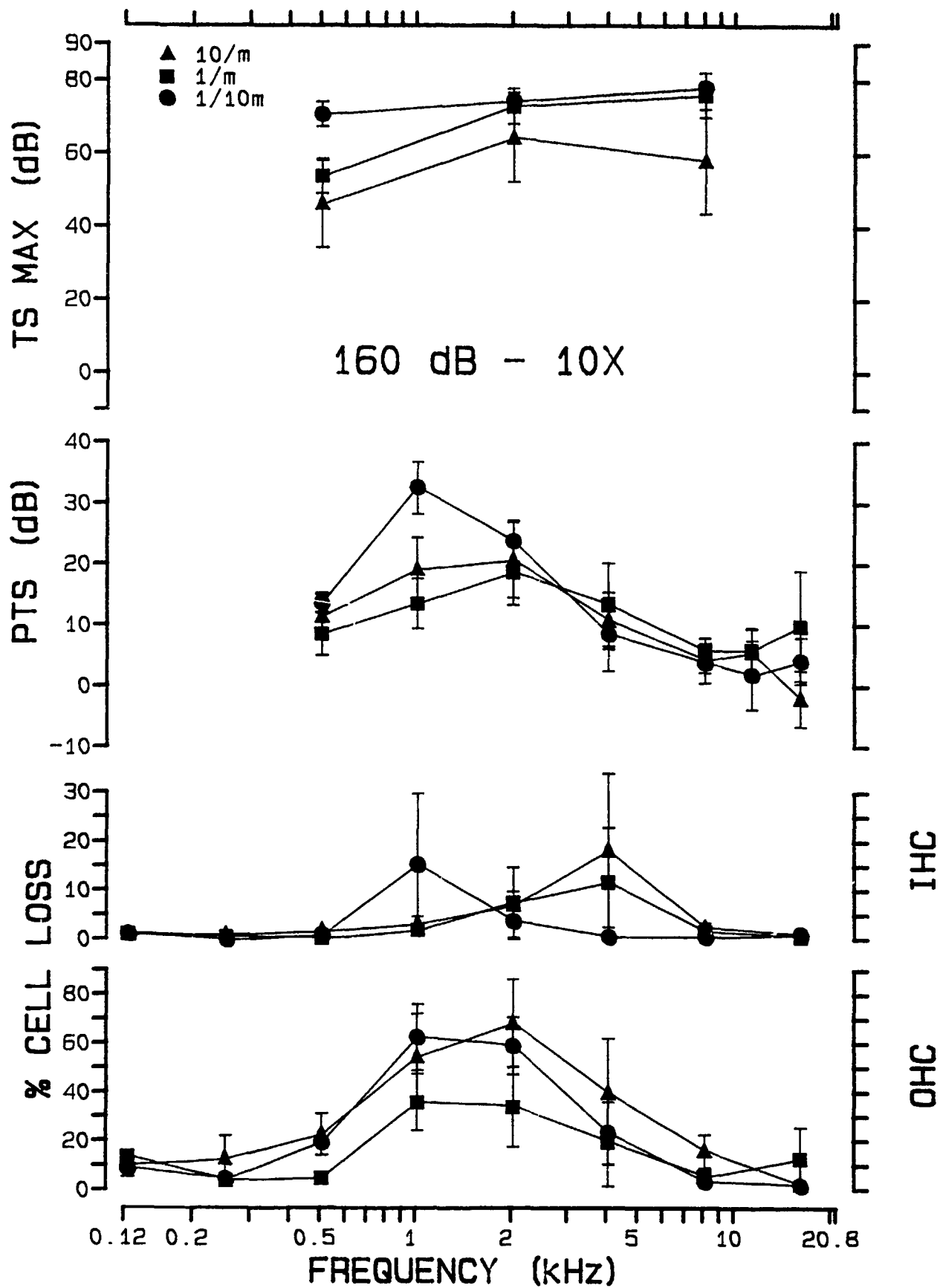
Figure 11. Mean TS$_{max}$ (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 160 dB peak SPL.
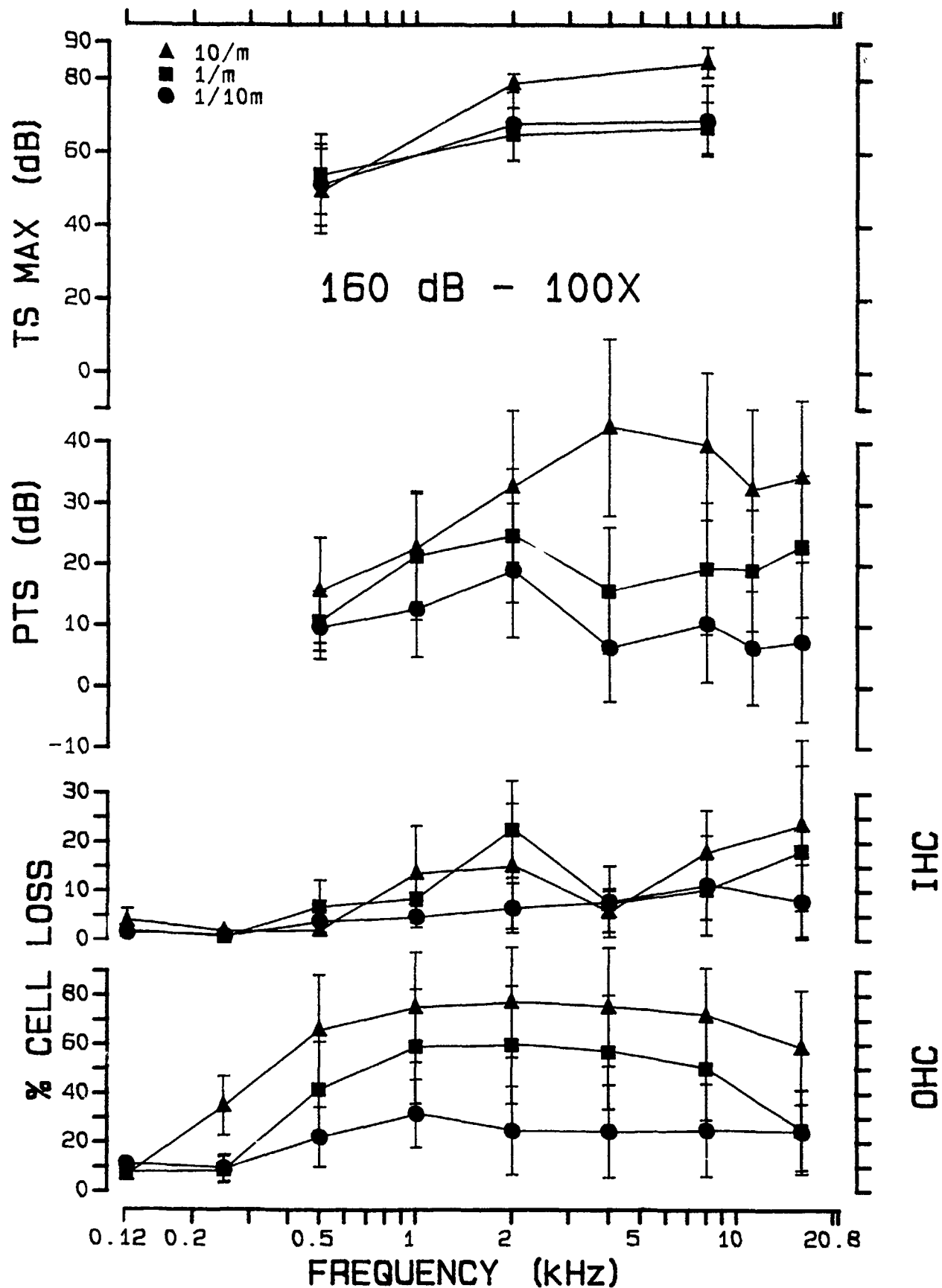
Figure 12. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 160 dB peak SPL.

subjects main effects will be dependent upon the audiometric test frequency or the location in the cochlea.

2. Impulse Presentation Rate: The effect of rate was examined in two separate analyses (Tables VIII & IX) of the groups exposed to 10 impulses and those exposed to 100 impulses. There were no statistically significant main effects of impulse presentation rate in any of the eight analyses.

There were significant interactions between presentation rate and frequency. A significant interaction between rate and frequency in the percent outer hair cell losses in groups exposed to 100 impulses suggested that the faster rates (10/m) were more hazardous than the slower rates. However, a parallel analyses of the groups exposed to 10 impulses showed a different pattern of interactions. When examining these groups, it was apparent that the groups exposed to the intermediate rate of one impulse per minute showed the greatest amount of damage at 155 dB but the least damage overall at 160 dB. Thus, in general a systematic or consistent effect of impulse presentation rate could not be extracted from these data.

In a report by Hamernik, et al. (1988), data resulting from a series of experiments similar to those reported here showed that using impulses that had a much lower frequency spectral distribution of energy, there was not a clear and consistent effect of impulse presentation rate upon the dependent variables. Tables XI and XII summarize the effects of impulse presentation rate on hearing trauma. This summary is derived from the data of Hamernik, et al. (1988) and this current report. Table XI represents a subjective decision based upon the plotted postexposure data concerning which of the three rates caused the most hearing loss or cell loss. The term "mixed" indicates an exposure for which no clear determination of the most hazardous exposure could be made. Multiple contrasts may be performed on individual means to determine which groups showed the greatest losses at individual frequencies. However, visual inspection of Figures 6 through 12 also provides an indication of which groups are most severely damaged by the impulse noise exposures. The conclusions made from the visual inspection are unlikely to be appreciably different than those made using a large number of multiple contrasts.

Table XI

Summary evaluation of the exposure producing the greatest degree of trauma based upon the repetition rate of the impulse. Trauma is estimated on the basis of PTS or sensory cell loss.

	Source I	Source II
150 dB 10X	mixed	mixed
150 dB 100X	1/m	mixed
155 dB 10X	1/m	1/m
155 dB 100X	mixed	10/m
160 dB 10X	10/m	mixed
160 dB 100X	10/m	10/m

A more objective approach to determining which exposure rate causes the greatest trauma can be obtained by computing the mean PTS evaluated at 1, 2 and 4 kHz and comparing the means across the various groups exposed to

different impulse rates. Table XII presents the summary of such an evaluation.

```
                              Table XII

   Summary evaluation of the exposure producing the largest mean PTS
       evaluated at 1, 2 and 4 kHz, based upon repetition rate of the
                               impulse

                         Source I          Source II

        150 dB 10X        1/m               1/10m
        150 dB 100X       1/m               10/m
        155 dB 10X        1/m               1/m
        155 dB 100X       10/m              10/m
        160 dB 10X        10/m              1/10m
        160 dB 100X       10/m              10/m
```

The results shown in Table XI and XII differ for some exposure conditions because the sensory cell losses did not necessarily correlate perfectly with the PTS measure. Thus, based on the data from 12 groups of animals that were exposed to blast waves at different rates, we are still unable to make any conclusive statement regarding the systematic effects of rate on the hazard to hearing resulting from blast wave exposure.

3. **Number of Impulses**: On the basis of the inconsistent effects of repetition rate on the dependent variables, we have collapsed the data across the rate variable to allow us to analyze the effects of number of impulses (since rate cannot be used as a variable for groups exposed to a single blast wave). By collapsing across rate, an analysis of variance can be performed using impulse peak pressure, number of impulses and frequency as the three main factors. In this analysis (see Table X), the main effect of number of impulses was statistically significant for both audiometric and both histological variables. Examining the figures it is clear that the single impulses caused the least amount of hearing and hair cell losses while the 100 impulses caused the greatest losses. The interaction of number of impulses and peak level was statistically significant for only the TS_{max} dependent variable. Thus, only for this dependent variable, the effect of peak or number was dependent upon the level of the other variable. In this case, there appears to be no effect of impulse peak for the groups exposed to a single impulse, but an appreciable effect of peak between groups exposed to 10 impulses. The interaction between impulse peak and number was not statistically significant for PTS or either of the histological variables.

4. **Impulse Peak Pressure**: The main effect of impulse peak pressure was a factor in each of the four analyses reported above. The results of these analyses were consistent in that, in general, with the same number of impulses, the 150 dB impulses were less hazardous than the 160 dB impulses, with the 155 dB impulses causing a somewhat intermediate effect on the dependent variables. In the analysis which included impulse peak and number (Table X), the main effect of peak pressure was statistically significant for all but the percent inner hair cell losses. Inner hair cell losses rarely exceeded 30% in any octave band region even in the most severe exposures. Therefore, it is likely that within-groups variability was simply too large relative to the between-groups variance to result in a statistically significant F ratio (F = 3.00, *df* = 2/95, p < 0.06).

The analyses of groups exposed to the same number of impulses show a similar effect of peak. In the groups exposed to a single impulse, a statistically significant interaction between peak and frequency for the TS_{max} dependent variable is illustrated by a higher TS_{max} in the 160 dB group than in the other two groups at the 2 kHz test frequency. Note that the peak of the A-weighted spectrum of the impact is at 1 kHz and should show its maximum effect one-half to one octave above that peak. The main effect of peak pressure was statistically significant for seven of the other eight analyses summarized in Tables VIII and IX. Once again, the percent inner hair cell losses did not show a statistically significant effect for groups exposed to 10 impulses. The effect of impulse peak pressure was statistically significant for all other dependent variables (i.e., TS_{max}, PTS, %OHC) and groups (i.e., 100X). Thus, as one would expect, the higher peak pressure impulses caused more damage than did the lower peak pressure exposures.

An alternate presentation of these data is shown in Figure 13 where the mean PTS evaluated at 1, 2 and 4 kHz ($PTS_{1,2,4}$) and the total inner and outer hair cell loss for each animal are shown plotted as a function of the total A-weighted sound exposure level. The sound exposure level is defined as:

$$10 \log_{10} \left[\frac{\int p^2(t)\,dt}{P_{ref}^2 \, \Delta t_{ref}} \right] \text{ where } p_{ref} = 20\mu Pa \text{ and}$$

$$\Delta t_{ref} = 1s.$$

The density of the data points makes it difficult to distinguish between the individual animals of each exposure group. Nevertheless, the presentation of data in Figure 13 clearly shows the increase in variability that occurs from exposure levels above about 125 dB. Consider, for example, the animals exposed to the more severe conditions (i.e., to A-weighted sound exposure levels above 140 dB). A number of animals show no PTS or sensory cell loss while others are severely traumatized. This degree of variability is a common observation following blast wave exposures and points out the need for an alternate approach to the data analysis. The second half of this report focuses upon one such alternate analysis.

C.   CONCLUSIONS

The following preliminary conclusions can be made from these data. (1) There was very little or no hearing loss or sensory cell loss for exposure to a single impulse at 150, 155 or 160 dB peak SPL. The variability among animals in these three groups was also relatively small. (2) There is a considerable increase in the variability or degree of susceptibility to trauma across animals as the severity of the exposure increases. This increase in susceptibility seems to be related more to the peak levels of the stimulus than it is to the total energy (Figure 13). The variability produces, in some extreme cases, a complete dichotomy in the results (i.e., within an experimental group of five animals, half the animals can show little or no effect of the exposure, while the remaining animals can be severely traumatized). Such a dichotomy makes it difficult to describe the data with conventional statistics. The only alternative seems to be to substantially increase the total number of animals in such exposure conditions. (3) With the above in mind a general, though not surprising trend in the data, is that as the peak levels and total energy increase permanent effects increase. These permanent effects seem to be dependent upon peak levels more than upon the total energy in the exposure stimulus (compare, for example, groups 6, 7 and 8 with groups 16, 17 and 18).

Symbol Key

●	150 dB 1X	■	155 dB 100X 10/m
◆	150 dB 10X 10/m	○	155 dB 100X 1/m
◄	150 dB 10X 1/m	◇	155 dB 100X 1/10m
¤	150 dB 10X 1/10m	◁	160 dB 1X
✚	150 dB 100X 10/m	×	160 dB 10X 10/m
☆	150 dB 100X 1/m	✖	160 dB 10X 1/m
▽	150 dB 100X 1/10m	✛	160 dB 10X 1/10m
△	155 dB 1X	►	160 dB 100X 10/m
✻	155 dB 10X 10/m	▼	160 dB 100X 1/m
▷	155 dB 10X 1/m	▲	160 dB 100X 1/10m
□	155 dB 10X 1/10m		
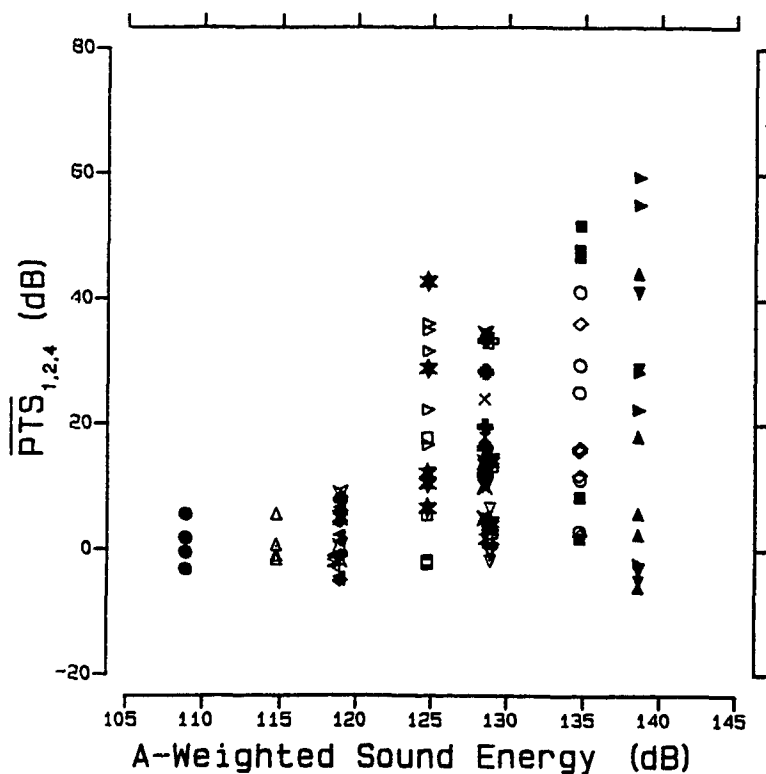
Figure 13. The distribution of mean $PTS_{1,2,4}$ and percent cell losses from individual animals (n=105) for all exposure conditions.
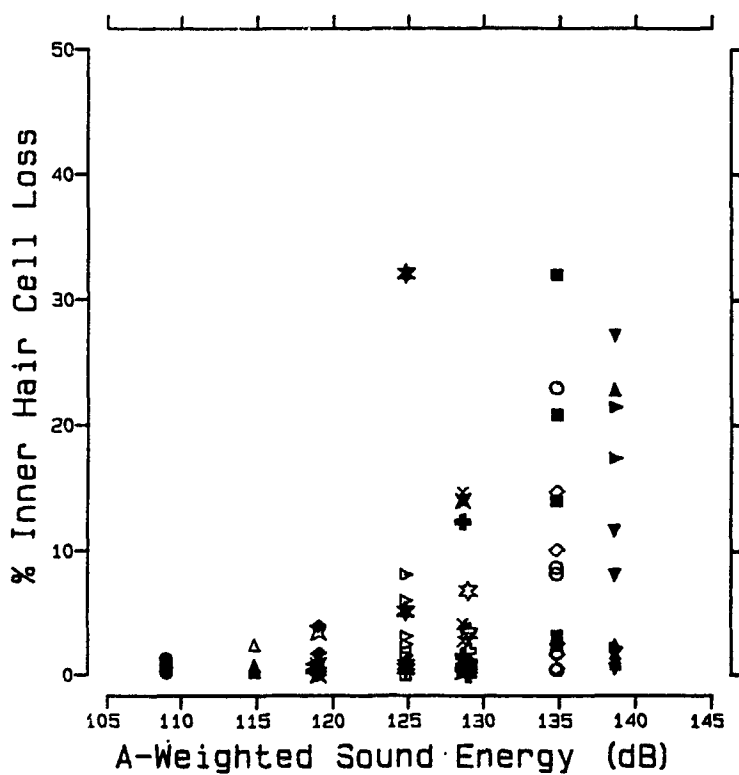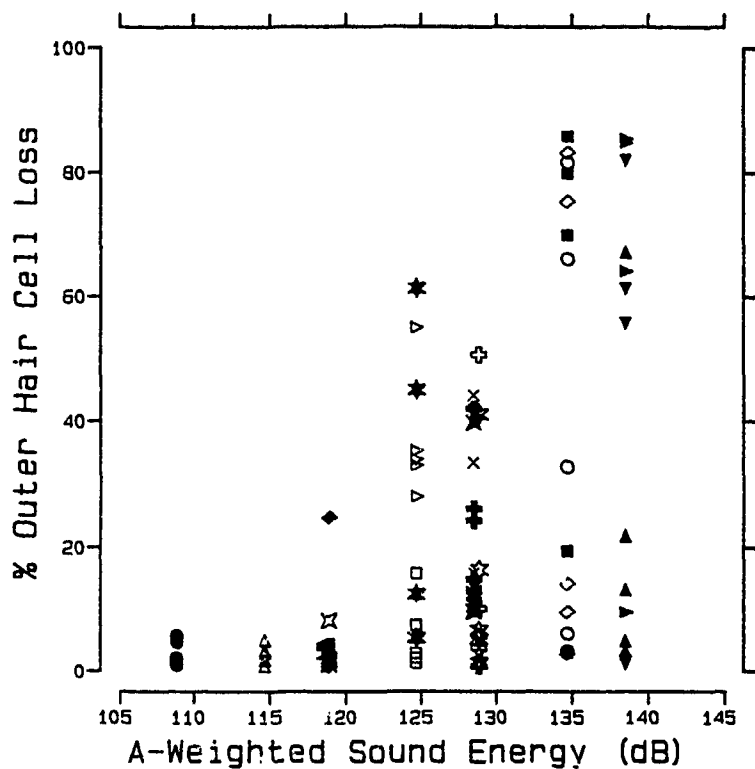
Figure 13. The distribution of mean $PTS_{1,2,4}$ and percent cell losses from individual animals (n=105) for all exposure conditions.

IV. Effect of Impulse Spectrum - Preliminary Analysis of Sources I and II

A.   METHODS

The data that are discussed in this section were acquired from a population of 214 chinchillas (109 exposed to Source I and 105 to Source II) that had a variety of impulse noise exposures. The impulses had A-weighted spectra that had two different distributions of energy that peaked at two different frequencies. Figure 14 illustrates the octave band energies for Source I and Source II.

Our intention in this section is not to present a set of conclusive results, but rather to illustrate an approach to the analysis of this type of experimental data that appears to be somewhat different from that which has been attempted in the past. It is an approach which develops a direct relation between frequency specific measures of PTS and the frequency domain representation of the impulse. The results of this approach can be directly related to the Price (1983) model and can also be used to estimate the permanent effects of a traumatic impulse noise exposure in a manner similar to that proposed by Kryter (1970) for estimating TTS after an impulse noise exposure. When data collection from all four sources is complete then an analysis of the complete data set following the ideas outlined below may clarify the relation between hearing trauma and the spectral characteristics of a nonreveberant impulse noise exposure.

PTS-Energy Flux Correlations: One of the problems that characterizes the measurement of PTS following impulse noise exposures is that of extreme intersubject variability. A number of authors have commented on this problem in the past, e.g., Kryter and Garinther, (1965), Henderson and Hamernik, (1982). The solution to this problem is to increase the number of subjects and thus increase the statistical power. However, the nature of these experiments is such that an excessively long time is required in order to run an experimental animal through a complete experimental paradigm of audiometric and histological protocols. This time factor effectively limits the number of animals in each experimental group and hence the statistical power. On the basis of a preliminary analysis of the PTS data produced in animals exposed to the two sources (see page 34), it was apparent that the effects on PTS of the different impact presentation rates showed, at best, an inconsistent pattern of results. Thus, a decision was made to evaluate all the PTS data without regard for presentation rate. Also, since relations between PTS and the increasing energy of the stimulus were being sought, presentation rate did not affect the independent variable. This effectively increased the number of animals at each sound exposure level to 15 except for the 1X exposure conditions. Total sound exposure or exposure level is increased by increasing the peak SPL or the number of impulse presentations.

For each audiometric test frequency a plot of the individual animal PTS at that frequency as a function of the total unweighted energy flux of the exposure in the octave band centered on that test frequency was prepared for each source. Two typical examples of this analysis at 2 kHz and 4 kHz for Source II are shown in Figures 15(a) and (b). For impact Sources I and II, 109 and 105 individual data points respectively were plotted for each source and for each frequency over approximately a 30 dB range of sound exposure level. The actual number of data points in Figures 15(a) and (b) is less than 105 since a number of animals had the same data coordinate. Using data sets such as those shown in Figures 15(a) and (b) the 90th percentile hearing loss (PTS$_{90}$) was computed for each sound exposure level at each octave frequency
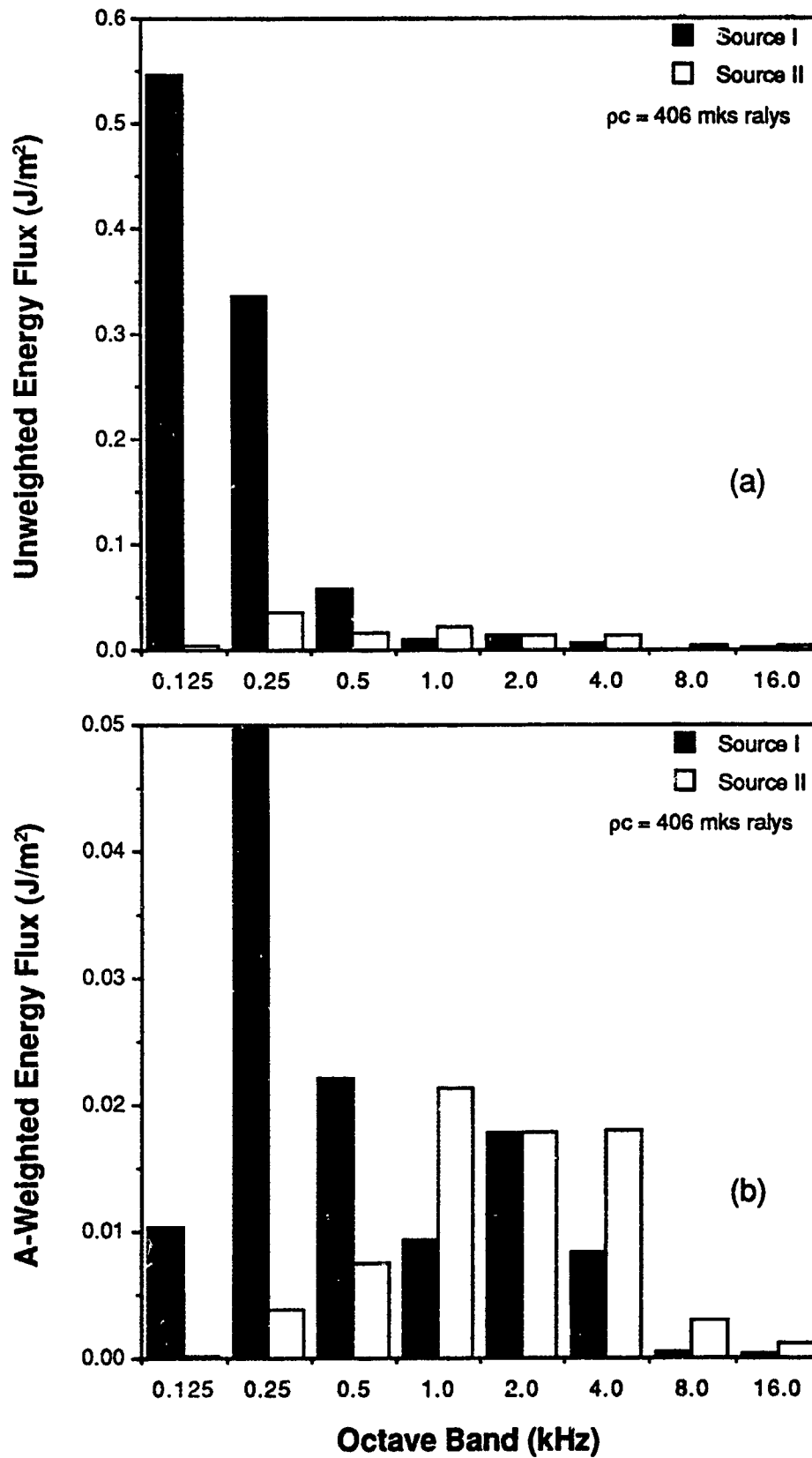
150 dB Peak SPL

Figure 14. (a-b) A-weighted and unweighted octave band spectra for the impulses produced by Source I and Source II at 150 dB peak SPL.
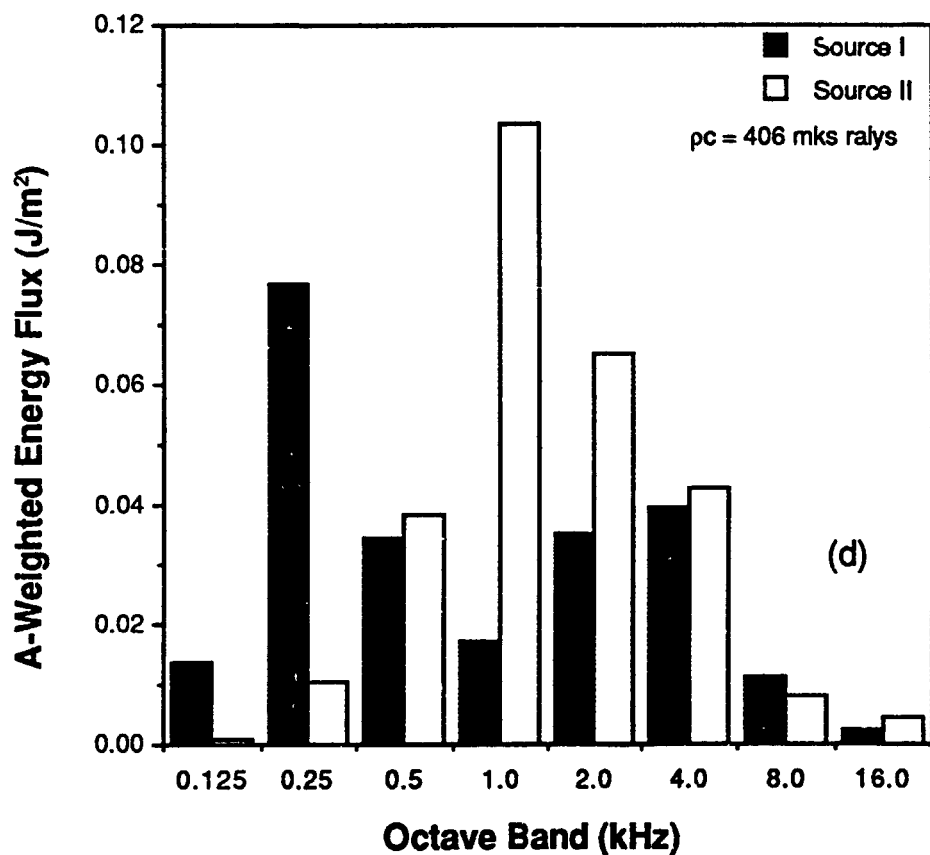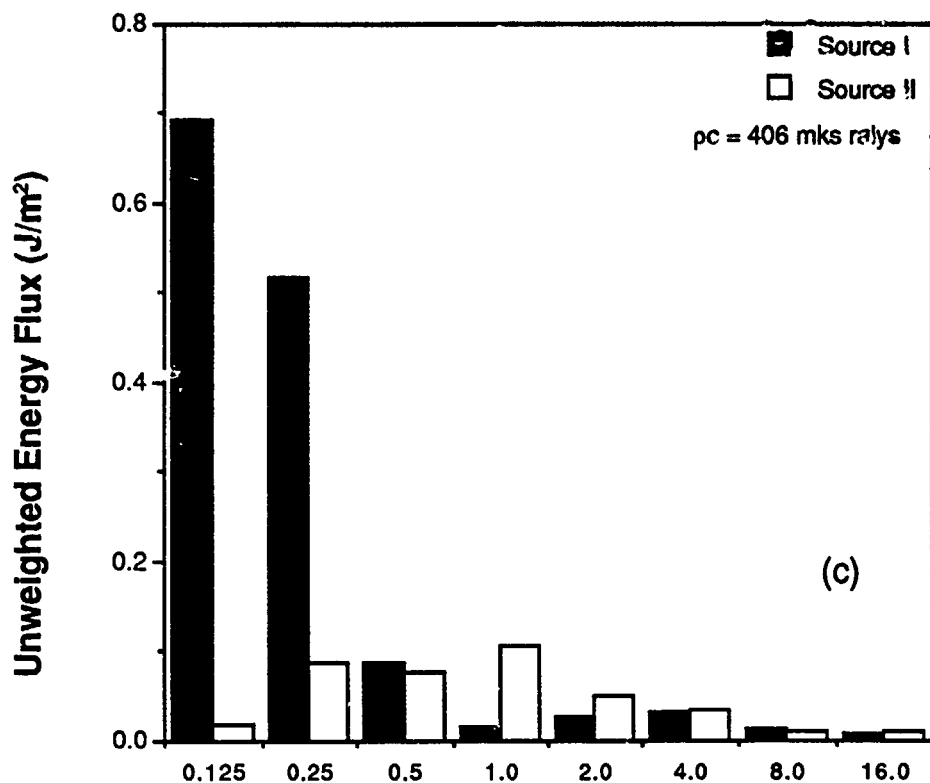
155 dB Peak SPL

Figure 14. (c-d) A-weighted and unweighted octave band spectra for the impulses produced by Source I and Source II at 155 dB peak SPL.
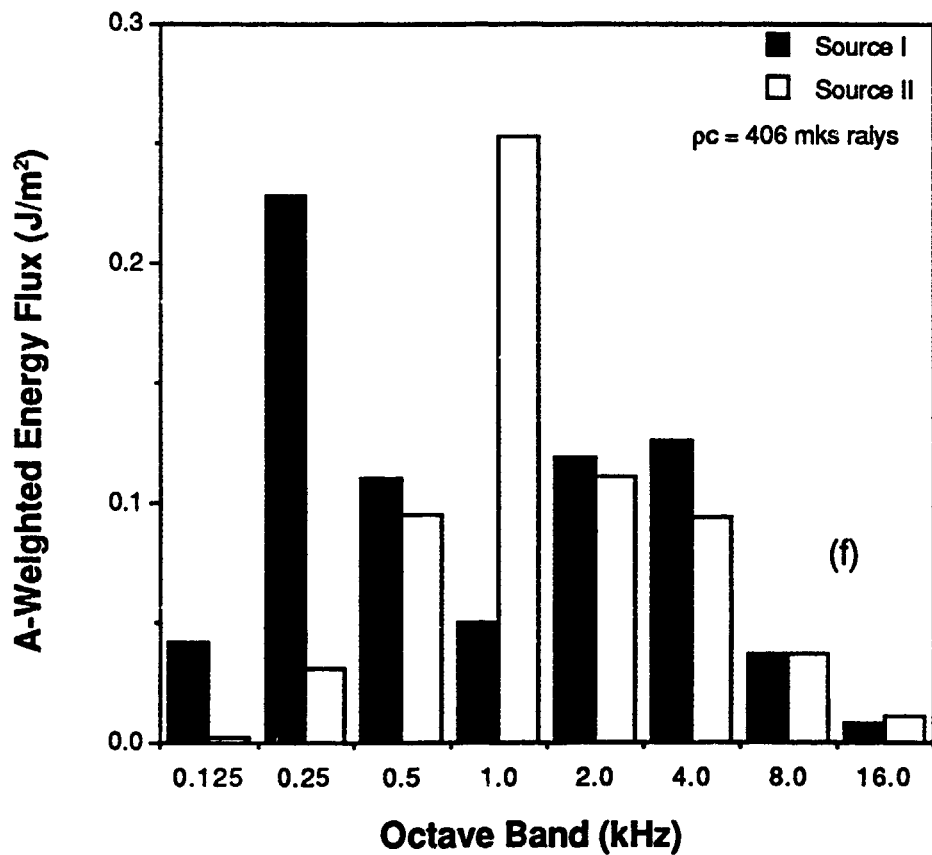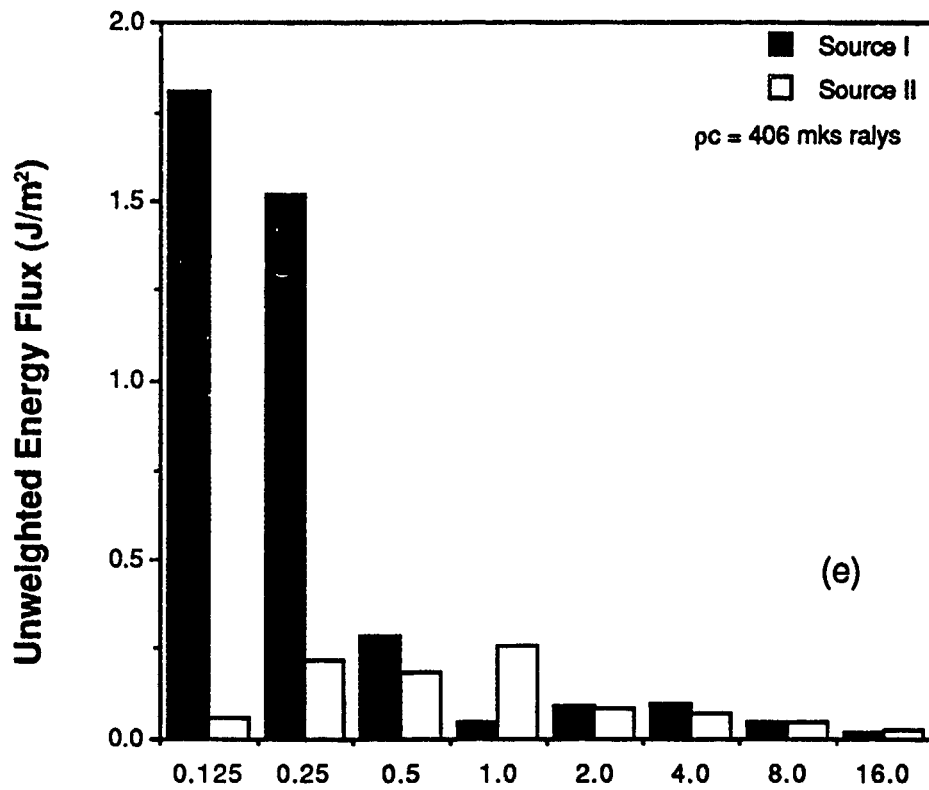
160 dB Peak SPL

Figure 14. (e-f) A-weighted and unweighted octave band spectra for the impulses produced by Source I and Source II at 160 dB peak SPL.
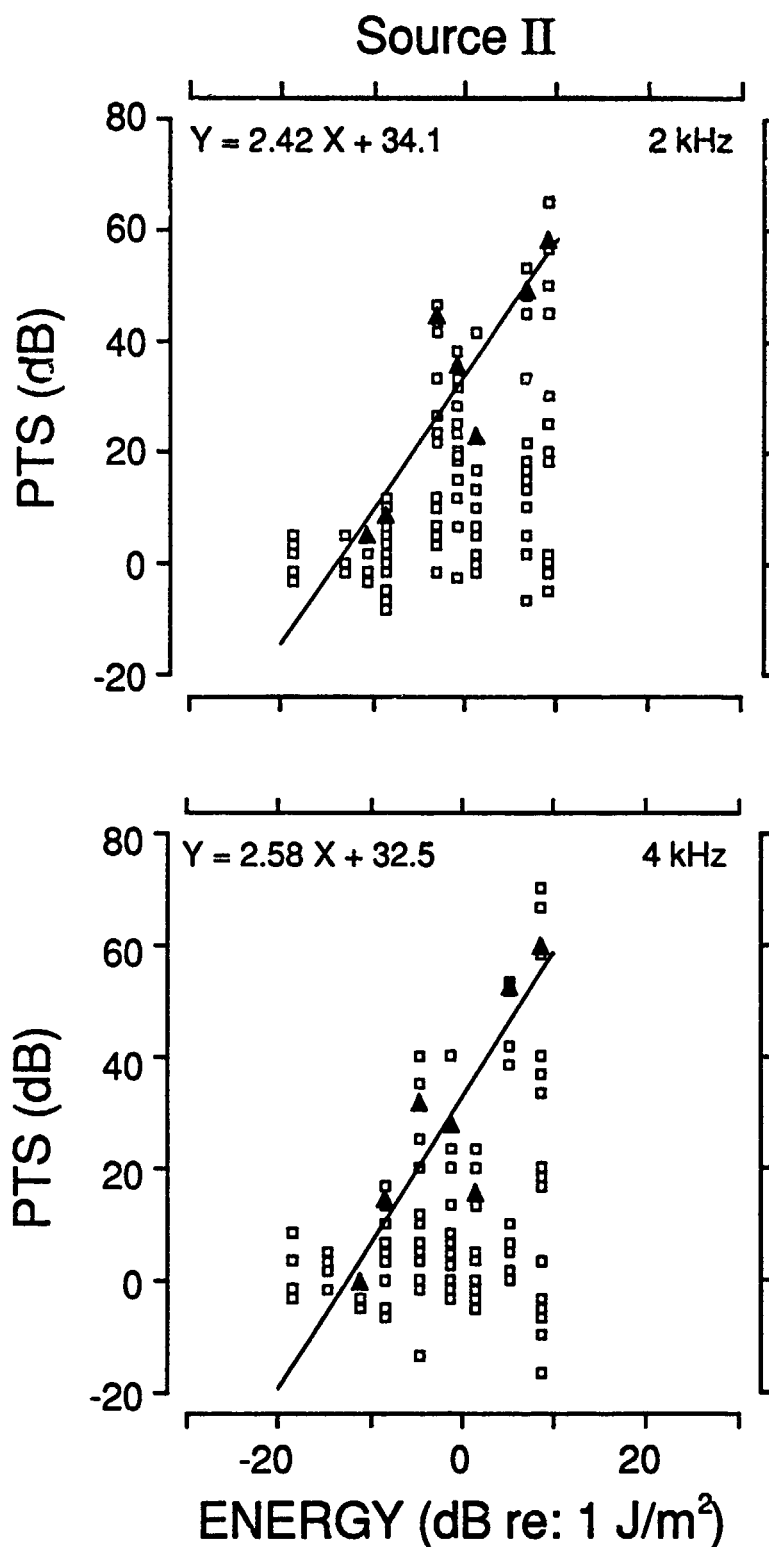
Figure 15. Two examples of the data analysis procedures at 2 and 4 kHz for Source II. The values of PTS for each animal are plotted as a function of the relative energy level in the octave band centered at the 2 and 4 kHz audiometric test frequency. The seven solid symbols represent the values of PTS_{90} which were used to obtain the regression line (solid line).

from 0.5 to 16 kHz. The 90th percentile, PTS_{90} at any frequency was computed from:

$$PTS_{90} = \bar{X} + st_{.10}$$

where, $\bar{X}$ is the group mean PTS; $t_{.10}$ is the value of the Student t distribution below which 90% of the PTS data lies; s is the group standard deviation.

This procedure yields a maximum of 9 percentile points shown by the filled symbols in Figures 15(a) and (b), i.e. three peak levels x three numbers of impacts. A linear regression line using these nine points was then computed. In many cases groups of animals exposed to only a single impulse did not show a PTS statistically different from zero at some frequency, and including these points in the least squares regression would artifactually decrease the slope of the regression line. In these cases only the highest energy group for which a PTS was not significantly greater than zero was used in computing the regression line. Thus, the regression line could be computed on a minimum of seven or a maximum of nine points. This exercise was repeated for each of the six octave test frequencies and for each of the two sources. From this set of regression lines the following relations could be obtained: (1) the threshold sound exposure level, $E_0(f)$, at each frequency for which 90% of the exposed population will have no PTS (i.e., the x-intercept of the regression line); and (2) the rate at which PTS_{90} increases as the sound exposure level is increased above the threshold level at each frequency (i.e., the slope of the regression line). Two other useful relations that can be obtained from (1) and (2) above are: the total sound exposure level in a given octave band required to produce a criterion level of PTS_{90} at that octave band center frequency or conversely the PTS that will not be exceeded by 90 percent of the exposed population for a specified exposure level (or energy level) in each octave band.

B.   RESULTS AND DISCUSSION:

A comparison between the impulses produced by the two sources at each of the three peak levels is shown in Figures 4 and 16. Along with the time waveform, the spectrum, $|P(\omega)^2|/\rho c$, of p(t) is also displayed. An analysis window of 33 ms was used in calculating the Fourier spectrum and ρc was taken as 406 mks rayls. The energy flux reference level for each spectrum is included in the graph. The narrow band spectra were also converted to an octave band format with center frequencies at the audiometric test frequencies in order to facilitate comparisons among impulses. The A-weighted and unweighted octave band display is shown in Figure 14. Because of the high levels of low frequency energy in Source I, the unweighted data lack the resolution at higher frequencies and comparisons between the two sources are difficult. However, after A-weighting, comparisons of the relative spectral distribution of energy across the two sources for different peak SPL is easily accomplished. For Source I, A-weighted energy is maximum in the 0.25 kHz octave band while Source II, it is a maximum at the 1.0 kHz octave band.

The results of the PTS-energy analysis described in the previous section are summarized in Figures 17(a) and (b), which illustrate the slope of the regression line and the intercept of this line with the abscissa. That is, the figures illustrate: (1) how hearing loss grows with increasing stimulus energy across the range of audible frequencies for the two classes of impulses; and (2) the threshold energy, $E_0(f)$, below which there should be no
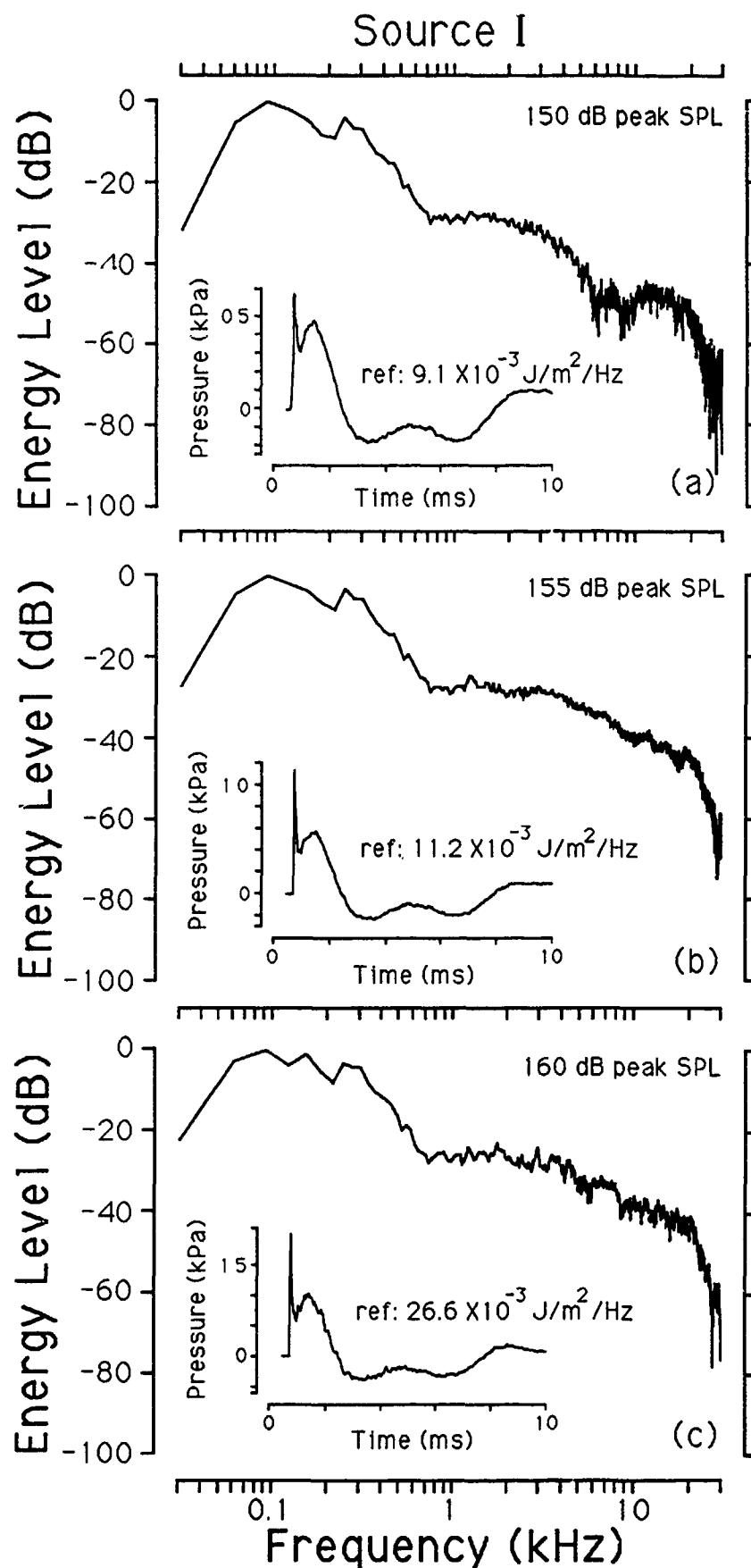
Source I

Figure 16.   (a)-(c): The spectra and the time waveforms for Source I at the three peak SPLs' that were used. The p(t) is shown for a 10 ms window while the spectra are calculated over a 33 ms window.
ρc = 406 mks rayls; f_s = 125 Hz; N = 4096; Δf = 30.5 Hz.

PTS incurred in 90% of the exposed population. The following generalizations can be made from this figure: (1) at a given audiometric test frequency, hearing loss grows at a rate of between approximately 1 and 3 dB of PTS for each dB increase in stimulus energy in that particular octave band; (2) the general trend for Source II is for the higher frequencies to have slightly higher growth rates; for Source I the function is fairly flat; (3) Source I, which produces the impulse whose spectrum peaks at the lowest frequency (.25 kHz) shows the lowest growth rates while Source II with the higher spectral peak (1 kHz) shows higher growth rates at the higher frequencies; (4) Source I and II both have a similar threshold energy, $E_0(f)$, for frequencies at and below 4 kHz while Source II has the higher threshold energy above 4 kHz.

Using the data embodied in Figures 17(a) and (b) the amount of PTS at each test frequency that will not be exceeded by 90% of the exposed population, for a fixed amount of energy, can be estimated. The result of this exercise is shown in Figure 18(a) for a criterion energy flux of 1 J/m^2. The data in this figure indicate that: (1) the level of PTS generally increases with increasing frequency; and that (2) Source I generally produces the lesser amount of PTS while Source II the greatest amount of PTS at the higher test frequencies. This figure also suggests that all energy is not equal in its ability to produce a PTS. At frequencies above 2 kHz, a 1 J/m^2 energy flux from Source II consistently produces more PTS than the same energy flux from Source I. An alternate way of stating this observation is that the energy, in a particular octave band, transported by an impulse whose spectral peak is at the very low end of the spectrum is less effective in producing a PTS than is the same amount of energy in the same octave band, transported by an impulse whose spectrum peaks at a higher frequency. Figure 18(b) illustrates the energy conveyed by an impulse necessary to produce a criterion PTS of 20 dB across the range of audiometric test frequencies. The functions for each of the two sources are similar across frequency. One of the surprising features of the data presented in Figures 17 and 18 is the general absence of a "most sensitive" frequency in the sense indicated by Price (1983). Rather, the general impression is that there is an approximately linear increase in susceptibility to NIPTS from the lowest to the highest audiometric frequencies.

For a point of comparison, it should be noted that Source I and II of this report produce impulses very similar to those produced by the 105-mm howitzer and the M-16 rifle respectively, as reported by Price (1983). In the Price experiments, while the cat was used as the animal model, the conditions of the exposure were a subset of those described in this report. Although the audiometric techniques differed, the distribution of individual animal PTS and the degree of variability are similar. However, because of the the differences in the manner in which the data are presented, detailed comparisons between the two studies are difficult to make.

While the above presentation of data for two different sources neglects some of the known characteristics of noise-induced hearing loss such as the half-octave shift, it is an approach that can be used to place bounds on the amount of PTS that can be expected from an impulse noise exposure on the basis of the spectral distribution of the energy of the exposure. If such an analysis is performed on a sufficiently large body of PTS data obtained from sources which cover the range of audibility in their spectral peaks, stimulus dependent bounds on PTS can be established for the chinchilla. Such an approach would be quite similar to that suggested by Kryter (1970) except that PTS rather than some measure of TTS would be predicted.
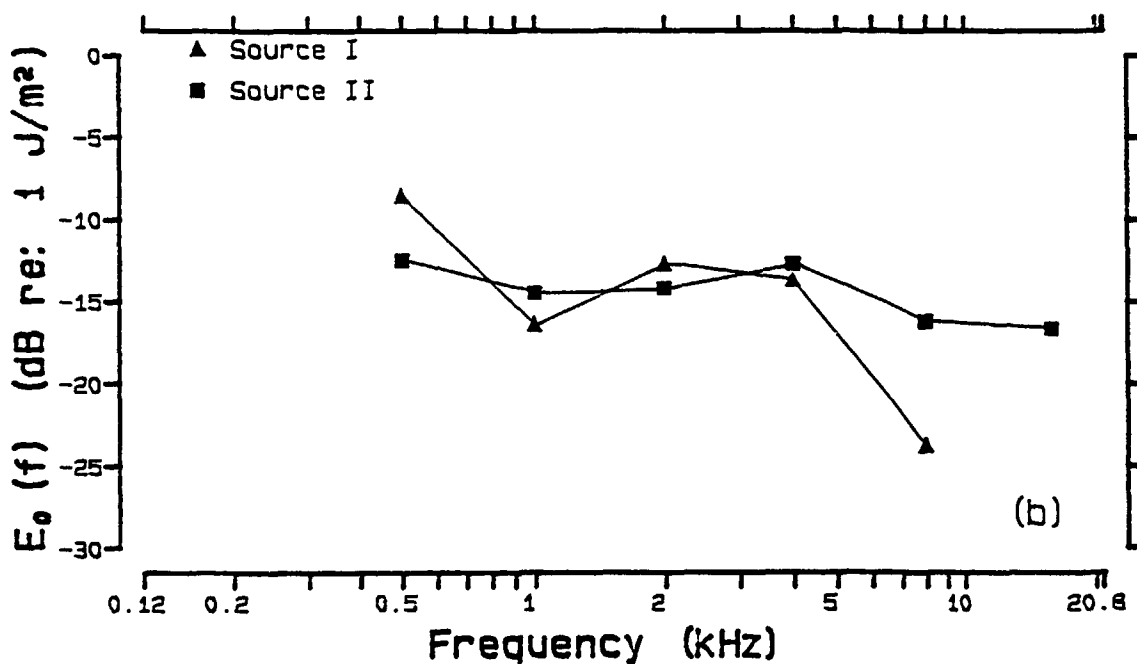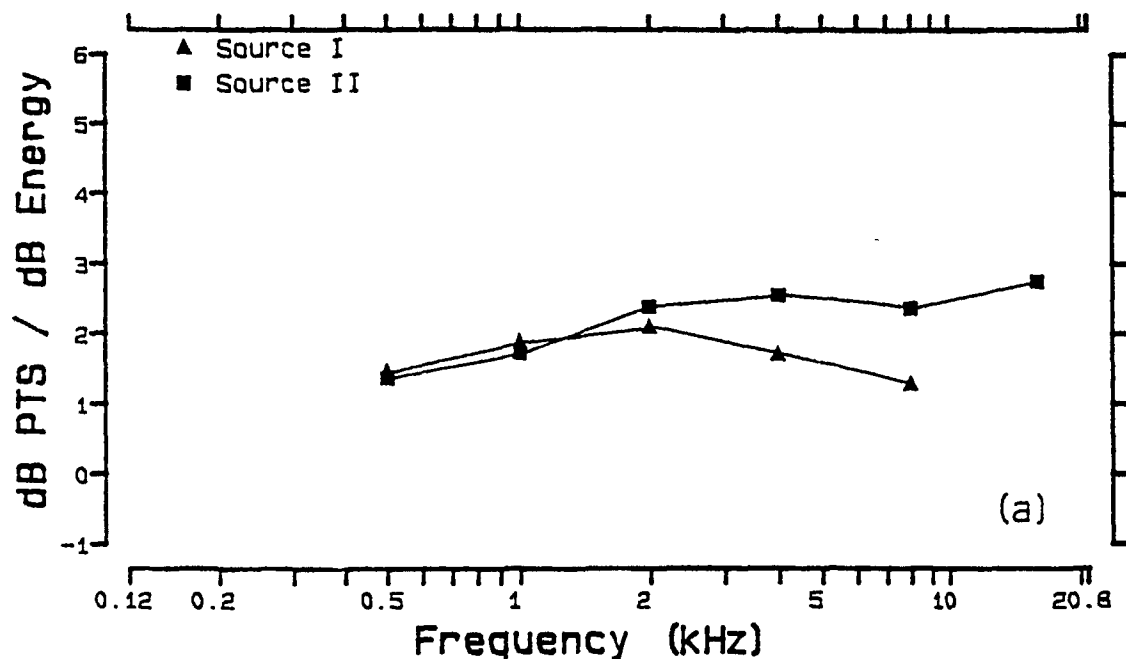
Figure 17. (a) The slope of the regression line relating PTS$_{90}$ and the exposure energy level across the range of audiometric test frequencies for the two impulse noise sources. (b) The threshold energy level E$_0$(f) at each audiometric test frequency and for each impulse source for which 90 percent of the exposed population will not sustain a PTS.
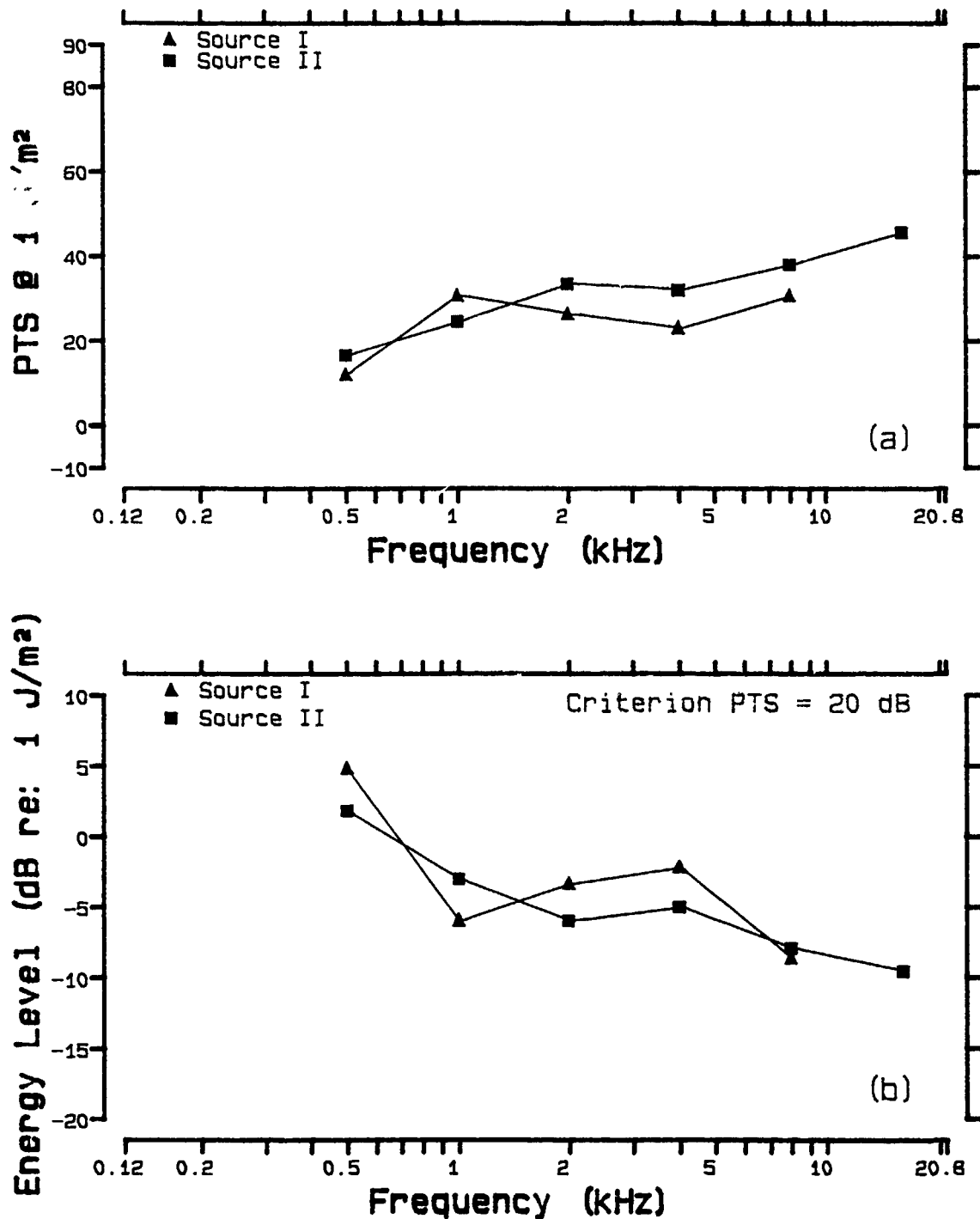
Figure 18. (a) The PTS_{90} that can be expected at each audiometric test frequency for an impulse having a 1 J/m^2 energy flux in each of the octave bands centered on the audiometric test frequencies. (b) The energy level in the octave band centered at each audiometric test frequency that will produce a criterion PTS_{90} of 20 dB.

The same approach to data analysis used in this present report can also be performed using the sensory cell loss in octave band lengths across the basilar membrane as the dependent variable. A criteria to limit or evaluate impulse noise exposures could thus also be established in terms of the potential for causing some criterion amount of sensory cell loss. Ideally, the predictions based upon the PTS and sensory cell loss analysis should be consistent with each other. The sensory cell loss analysis is still in progress.

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